THE HISTORY of GENERAL PARALYSIS of the INSANE in BRITAIN, 1830 to 1950

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Abstract of Thesis

Juliet D Hurn, The History of General Paralysis of the Insane in Britain, 1830 to 1950

This thesis explores the history of ideas about, and responses to, general paralysis of the insane (GPI) - specifically in the context of the developing profession of psychiatry in Britain. It considers GPI as an objective disease entity whose subjective definition was nevertheless open to negotiation; for example, in deciding how central was overt insanity, or how GPI should be differentiated from the allied disease of tabes dorsalis. It explores how psychiatrists' interest in organicism and the science of medicine - and their attempts to raise the status of their specialty - both informed their understanding of GPI, and allowed them to promote it as a flagship disease for their profession. Nevertheless it draws attention to the gap between such claims and concrete practical advances which the disease fostered. The thesis considers changing causal explanations for GPI: first, in relation to the evolving image of the typical general paralytic patient; and second, in relation to the credence attached to different forms of causal evidence such as pathology, statistics, and laboratory medicine. It suggests how assessment of this evidence might have been informed both by professional aspirations and by pervasive cultural concerns such as fear of syphilis and degeneration theory. The thesis studies the use of malaria therapy to treat GPI in Britain, and uses this episode to explore a number of themes: early twentieth century ethical attitudes to heroic treatments; perceptions of 'cure'; and the change in emphasis from asylum care to community care. Finally, it considers ideas about the epidemiological history of the illness - from early twentieth-century theories about the evolution of infections, to Edward Hare's hypothesis of a neurotropic epidemic; and considers how the views of disease as objective entity, and disease as cultural construct, might be reconciled in the context of GPI.

I would like to thank the numerous library staff who orientated me during my research, particularly those at the Wellcome Institute, the Greater London Record Office, and the Public Record Office at Kew. I am particularly grateful to those individuals who helped me to locate and use the more elusive archives of Horton and Claybury asylums. Thank you to those colleagues at the Wellcome who have offered practical advice over the past three years, to my 'office-mates' who have shared numerous laughs and cups of hot chocolate, and above all to my supervisor, Bill Bynum, for his ever-patient support, his constructive suggestions, and for his enthusiasm. Finally, my family and a number of close friends have been very important to me - particularly during the rather frustrating last stages of the thesis. I would like to thank them very much for their constant encouragement and for their kindness.

CONTENTS

Contents cntd

INTRODUCTION

In 1859 the medical superintendent of Bethnal House Asylum wrote a vivid description of a patient's descent into insanity and death:

'A person who is insane ... [shows] slight tremors of the lips ... and feeble, straddling, or devious [gait] ... He is full of all manner of schemes ... and talks of the wealth he fancies his projects have brought him ... The whirl of the spirits increases ... Arrived at this pitch, everything becomes invested with immensity, grandeur, or beauty. Common pebbles are transformed into gems ... [Thereafter] incessantly talking and restless, violent and destructive, tearing everything tearable to shreds ... he lies on his bed ... or on the padded floor of his room in a dream of happiness and splendour, which contrasts horribly with his hollow features and emaciated, squalid body. Happily death is at hand - exhaustion or paralytic coma soon closes the scene.'[1]

The patient in question was suffering from general paralysis of the insane (GPI), a disease which had captured the imagination of British doctors through a number of striking features. First, it was a form of insanity which had sprung to prominence only since the 1820's - appearing first in the mental hospitals of Paris and thereafter being recognised increasingly within English asylums. During the nineteenth century it would rise inexorably, so that by the end of the 1880's it would

[1] T J Austin, A Practical Account of General Paralysis, its Mental and Physical Symptoms, Statistics, Causes, Seat, and Treatment (London: Churchill, 1859); extract reproduced in R Hunter and

account for roughly 15% of annual male asylum admissions.[2] Although this was not a startling proportion, its clinical course and invariable fatality meant that it accounted for a far larger percentage of asylum deaths, and demanded intensive and exhausting nursing care. Furthermore, since it attacked predominantly middle-aged men, it was regarded as having particularly important socio-economic implications. Second, it followed a peculiarly grotesque and degrading clinical course. Although the emphasis attached to different symptoms would vary over time, the above portrayal was a typical one, contrasting as it did florid delusions of grandeur - in which the patient typically believed he owned everything around him - with a sordid physical decline ending in total paralysis, gangrene, and death. Third, notwithstanding the powerlessness of alienists to influence its course, GPI threw interesting light upon some of the most important intellectual and professional problems of mental medicine.

Considering its importance to nineteenth and early twentieth-century psychiatrists - not to mention the men and women afflicted by it - GPI has attracted surprisingly little attention amongst historians. Accounts of the disease have predominantly been in the triumphalist tradition, and have been short, even passing, references placed in the context of the march of medicine or psychiatry as a whole.[3]

I Macalpine, Three Hundred Years of Psychiatry 1535 - 1860 (London: Oxford University Press, 1963), pp 1053-7; on p 1056.

[2] The figures, for example, for 1887 were 1062 male admissions (compared with 7086 admissions as a whole); and 238 female admissions (compared with 7250 admissions as a whole).

[3] Here, and in future references, I use the term 'triumphalism' to mean a style of history-writing in which it is assumed that scientific progress can be charted through the approach towards an objective scientific truth. Such history-writing tends to be retrospective: present-day knowledge is taken as the goal towards which past generations have been striving. It also tends to be judgmental: praising the work of those perceived to have aided progress; and dismissing the work of those perceived to have hindered it. Although such triumphalism has been largely eschewed by historically-trained writers

Henry and Zilboorg, Ackerknecht, and Hunter and Macalpine, for example, have produced more or less detailed histories of this kind, chronologically tracing the discovery and clarification of the disorder by the scientific process.[4] Key points are emphasised in such accounts: the French alienist Bayle's first clear description of GPI, published in 1822; the elaboration of its clinical and pathological features; the French venereologist Fournier's championing of its syphilitic aetiology during the late nineteenth century; and the work of the laboratory scientists Noguchi and Moore who identified the syphilitic organism in the brains of general paralytics - thus vindicating Fournier's earlier arguments. Malaria therapy, used to combat the disease from the 1920's, has attracted some interest - although again far less than it deserves. This interest has again been primarily in the triumphalist mould; and latterly the episode has been used to critique pre-war experimental methods, by comparison with current standards of therapeutic efficacy.[5] Neither approach offers a fully contextualised account of the aims and implementation of this early 'heroic' physical treatment.

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since the 1960's, it remains the dominant approach amongst medically-trained writers. It is exemplified in the work of Hunter and Macalpine, psychiatrists who charted the progress of their specialty towards the goal of rigorous organicism: Hunter and Macalpine, 1963.

[4] G W Henry, 'Organic Mental Diseases' in G Zilboorg and G W Henry, A History of Medical Psychology (New York: W W Norton, 1941), pp 526 - 551; E Ackerknecht, A Short History of Psychiatry, transl S Wolff (New York & London: Hafner, 1959), pp 66-7; Hunter and Macalpine, 1963, pp 1052-3. See also F G Alexander and S T Selsnick, The History of Psychiatry (London: George Allen and Unwin Ltd, 1967). Although still in the 'progressive' mould, George Rosen and Crissey and Parish's accounts are more unusual in providing a fuller sociological context to the disease. G Rosen, Madness in Society: Chapters in the Historical Sociology of Mental Illness (New York: Harper & Row, 1968), pp 248 - 258; J T Crissey and L C Parish, Dermatology and Syphilology of the Nineteenth Century (New York: Praeger, 1981).

[5] J Purdon Martin, 'Conquest of General Paralysis', BMJ, 15 July, 1972, 3, pp 159 - 160; S C Austin, P D Stolley, T Lasky, 'The History of Malariotherapy for Neurosyphilis: Modern Parallels', JAMA, July 22/29, 1992, 268: 4, pp 516 - 519; H R Rollin, 'The Horton Malaria Laboratory, Epsom, Surrey (1925 - 1975), J Med Biog, May 1994, Vol 2, No 2, pp 94 - 97; M Whitrow, 'Wagner-Jauregg and Fever Therapy', Med Hist, 1990, 34, pp 294 - 310; Idem, Julius Wagner-Jauregg (1857 - 1940) (Nishimura: Smith-Gordon, 1993).

The traditional accounts take it for granted that, because GPI was quickly recognised as a structural disease of the brain with a predictable clinical pattern, it provided the supreme scientific model for psychiatry in a way which other mental diseases could not. This argument has served both those historians who applaud the model of organicism, and those who regard it as a regrettable step away from psychological or psychoanalytical conceptions of mental disease.[6] The latter assumption inspired Thomas Szasz, an anti-psychiatrist writing during the 1960's, to single out GPI as the bête-noire of his profession. The discovery of its syphilitic cause, he claimed, encouraged twentieth-century psychiatrists to model all mental diseases upon unrealistic medical ideals: 'Is it any wonder ... psychiatry speaks with the accents of neurosyphilis on its lips? ... Now while asleep, psychiatry still dreams about it; and while awake, it sees the world as if the spectre of paresis lurked behind every foolish face or troubled thought. Thus has the image of the crooked spirochaete [the syphilitic organism] making people mad been replaced, in the minds of many psychiatrists, by the image of the crooked molecule making them mad.'[7]

GPI, then, has all too often been viewed by historians as a disorder which must say something about modern medicine: as Rosenberg comments: 'Whether or not general paralysis ... is or should be a model for psychiatry ... remains an unresolved problem.'[8] Such a retrospective view suggests a rather uncritical approach to the question of exactly how GPI made its effect upon the contemporary profession.

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[6] To the first class belong the majority of the traditional accounts - epitomised by the historian-psychiatrists Hunter and Macalpine.

[7] T S Szasz, 'Schizophrenia: The Sacred Symbol of Psychiatry', Brit J Psychiatry, 1976, 129, pp 308 - 316; on p 309-10.

A handful of historians have made more incisive or varied references to the story of GPI, and interestingly the attention has come mainly from within the ranks of psychiatrists. The single most lengthy account of the disease was published in 1959 by Hare, and concerned the historical epidemiology of GPI: specifically the reasons for its apparently sudden appearance in the early nineteenth century, its subsequent rise and spread through Europe and America, and its decline from the 1920's onwards.[9] The account, which I discuss in the final chapter of this thesis, was a detailed piece of analytical work. Hare's aim, however, was not to write a comprehensive history of GPI and its contemporary meanings, but rather to support his strictly biological conception of the major mental diseases through his theory of infective aetiology and disease mutation over time.

Antoine Bayle, the putative discoverer of GPI, has attracted some attention - although primarily amongst French historians - mainly because he appeared to be ousted from the psychiatry profession shortly after his early work upon the disorder. Bercherie explains this on the basis that Bayle's description of GPI challenged the nosological scheme of the influential Esquirol, by depicting the disease as an unfolding clinical entity which cut across symptomatic elements.[10] Goldstein suggests simply that Bayle's mentor died, leaving the young alienist with no professional support.[11] E M Brown goes beyond Bercherie's thesis by claiming that

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[8] C E Rosenberg, 'The Crisis in Psychiatric Legitimacy', in G Kriegman et al (eds), American Psychiatry, Past, Present, and Future (Charlottesville: University of Virginia Press, 1975), pp 135-48.
[9] E H Hare, 'The Origin and Spread of Dementia Paralytica', J Ment Sci, 1959, 105, pp 594 - 626.
[10] P Bercherie, Les Fondements de la Clinique (Paris: La Bibliotèque d'Ornicar, 1980), Ch 5, esp pp 70-76.
[11] J Goldstein, Console and Classify: The French Psychiatric Profession in the Nineteenth Century (Cambridge: Cambridge University Press, 1987), pp 146-7.

Bayle's whole advocacy of the anatomo-pathological approach to mental disease threw itself against Pinellian principles.[12] Finally Berrios, a psychiatrist-cum-historian with a particular interest in the changing classification of mental disorders, moves beyond the early French history of the disease, and turns particularly to its epistemological interpretation during the nineteenth century.[13] Specifically he challenges the traditional contention that GPI reinforced alienists' belief in the anatomo-clinical view of mental disorder. In part this claim draws upon Bercherie's suggestion that the importance of Bayle's work lay less in its anatomical than in its natural historical conception of GPI. In part it springs from Berrios' belief that alienists were not in fact wedded to the ideals of organicism during the nineteenth century; that GPI was never a conceptually stable disease; and that the disorder 'drew the minds of alienists more towards psychological and philosophical issues than towards anatomy.'[14] This revisionist account of GPI is one that I shall refute on several counts.

Turning to a later period in GPI's history, Braslow discusses the implementation of malaria therapy in American state hospitals, arguing that it allowed the more active participation of patients in their treatment regimes, and a more empathic relationship between patients and doctors.[15] This is a perceptive and

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[12] E M Brown, 'French Psychiatry's Initial Reception of Bayle's Discovery of General Paresis of the Insane', Bull Hist Med, 1994, 68, pp 235 - 253.

[13] G E Berrios, ''Depressive Pseudodementia' or 'Melancholic Dementia': A Nineteenth Century View', J Neurol, Neurosurg, & Psychiatry, 1985, 48, pp 393 - 400; G Berrios and R Porter (eds), 'Dementia' in A History of Clinical Psychiatry: The Origin and History of Psychiatric Disorders (Falmouth: The Athlone Press, 1995), pp 38 - 40. The brevity of this account nevertheless demonstrates the relative lack of interest which GPI has attracted.

[14] Berrios, 1985, p 397.

[15] J T Braslow, 'Effect of Therapeutic Innovation on Perception of Disease and the Doctor-Patient Relationship: A History of General Paralysis of the Insane and Malaria Fever Therapy, 1910 - 1950',

original approach to the episode which avoids commenting upon whether or not malaria therapy 'worked'; although it could be argued to serve Braslow's (medically orientated) stance that biological therapies can enhance doctor-patient communication rather than hindering it.

These, then, are the major existing histories of GPI, and their dearth suggests that the disease deserves a fuller account. Writing the history of any disease is, of course, fraught with difficulties. Recent scholarship - particularly that informed by the sociology of knowledge - has stressed the concept of disease as a culturally-determined construct rather than a transhistorical biological entity.[16] In practice, such an approach has been far more extensively applied to 'mental' than to 'physical' diseases - since the history of psychiatry has been marked by obvious cultural ramifications and by profound classificatory shifts which have belied the concept of an underlying organic truth. Micale's enquiry into hysteria, for example, provides a persuasive argument for formulating a 'thoughtful historical epistemology' which acknowledges the interpretative and conceptual complexities of disease definition, rather than assuming biological or even linguistic continuity.[17] Yet Micale himself goes on to suggest that symptomatic consistency, or even biological 'truth', can make the historian's task simpler; and that this might apply, for example, to organic

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Am J Psychiatry, May 1995, 152: 5, pp 660 - 665; Idem, 'The Influence of a Biological Therapy on Physicians' Narratives and Interrogations: The Case of General Paralysis of the Insane and Malaria Fever Therapy, 1910 - 1950', Bull Hist Med, 1996, 70, pp 577 - 608.

[16] See, for example, C E Rosenberg and J Golden (eds), Framing Disease: Studies in Cultural History (New Brunswick, New Jersey: Rutgers University Press, 1992).

[17] M S Micale, Approaching Hysteria: Disease and Its Interpretations (Princeton: Princeton University Press, 1995).

diseases such as the infectious fevers.[18] Historians have indeed felt far more comfortable in viewing these kinds of diseases as transhistorical entities, writing about their retrospective epidemiology, and offering explanations based upon present-day knowledge. Difficulties of historical sources and past modes of classification have of course been addressed, but have not challenged the notion of an enduring object that can be scrutinised. Taking such transhistorical truth for granted, historians have then felt comfortable in turning their attention, for example, to social responses to these diseases.[19]

GPI lies, interestingly, between these two kinds of historical object. Although a psychiatric disease with strong cultural resonances, it has always been regarded primarily as an 'organic' syndrome. Indeed, this may be one reason for the surprising lack of interest shown in it by recent psychiatric historians: its undeniably concrete physical symptoms have perhaps made it seem less amenable to theories of social construction than its fellow psychiatric disorders.[20] In this thesis I shall argue that there were certainly significant changes in the conception of the disorder over the one hundred or so years of its prominence; changes which were inseparable from both professional interests and broader social movements. During the mid-nineteenth century arguments surrounded the relationship between, and relative necessity of,

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[18] 'For diseases with known organic aetiologies and clear and consistent symptom profiles, the epistemological conditions for historical enquiry will be simpler and less confining ...' Micale, 1995, p 114.

[19] Hence, for example, copious histories of tuberculosis: among them L Bryder, Below the Magic Mountain (Oxford: Clarendon Press, 1988); B Bates, Bargaining for Life: A Social History of TB, 1876 - 1938 (Philadelphia: University of Pennsylvania Press, 1992).

[20] Szasz, for example, turned his attention primarily to schizophrenia. Interestingly, Micale seems to ignore GPI when he claims that the depressive disorders are the only mental category which demonstrate transhistorical consistency of symptom profiles: Micale, 1995, p 114, referring to S W

mental and physical symptoms of the disease. Different names given to the disorder reflected this: 'general paralysis of the insane (GPI)', for example, was derived from an 1826 French term, and emphasised the integral part played by insanity - pace the looser term 'general paralysis'. Neurological techniques during the same period established that patients' gradual loss of limb use was not due to true muscular paralysis, but due to loss of the 'will' to move. 'Paralysis' thus seemed inappropriate to many doctors, and 'general paresis' - which became popular in America - was a suggested alternative which took its place beside a bewildering number of other names, including the German favourite 'dementia paralytica'.[21] 'GPI' was open to other objections, including the observation that 'one who is generally paralysed is almost certainly dead'; but this 'more familiar misnomer' continued to be the term most commonly used in Britain, and I have used it throughout my thesis.[22] Although insanity was rapidly established as central to the disorder, the depiction of the accompanying mental disturbance itself changed subtly over the course of a hundred years from an emphasis upon exuberant grandiose delusions to an emphasis upon gradual dementia and mental enfeeblement. These symptomatic perceptions contributed to tenacious broader images of the disease and of the general paralytic 'character', which themselves altered over the course of time. More emphatically, the application of the Wassermann reaction - a diagnostic test for syphilis - added a new

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Jackson, Melancholia and Depression: From Hippocratic Times to Modern Times (New Haven: Yale University Press, 1986).

[21] See Hare, 1959, who notes that the disease 'has suffered from a plurality of names', p 595, f/n 1. See Mickle's list of synonyms in W J Mickle, General Paralysis of the Insane, 2e (London: H K Lewis, 1886), p 1.

[22] J Wilkie Burman, 'A Contribution to the Statistics of General Paralysis', in J Crichton Browne (ed), The West Riding Lunatic Asylum Medical Reports, 1871, 1, pp 129 - 151; on p 129.

laboratory criterion to the identity of the disease during the early twentieth century. The term 'neurosyphilis' - a definition which embraced GPI, the spinal paralysis 'tabes dorsalis', and disorders symptomatically somewhere between the two - became the usual epithet, and emphasised both the new aetiological conception of GPI and the role of the laboratory in making the diagnosis. Finally, during the 1920's the dramatic new malaria treatment for GPI threw into question for the first time whether the disorder was necessarily fatal.

Given these changing interpretations of the disease, is it meaningful to follow the history of 'GPI' as though it were a conceptually stable entity? Berrios suggests that the definition of the disorder was very broad, and open to vast disagreement during the nineteenth century. Many cases diagnosed as such, he notes, subsequently recovered; leading him to the suspicion that they were confused with disorders such as depressive pseudo-dementia. In a particular context - that of case histories published in a book entitled Melancholic Dementia by the French psychiatrist Mairet - he suggests a marked narrowing in the clinical boundaries of GPI at the turn of the century as dementia itself was re-defined. Such fluidity, he claims, belies the notion that GPI might have been used as a scientific 'model' of mental disease by nineteenth century alienists.

This problem of clinical definition was one of which alienists were certainly well aware.[23] Nonetheless, I would maintain that GPI did indeed maintain relative conceptual stability even as doctors' images and interpretations of it changed. This

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[23] See, for example, Mickle's discussion of the problems in delimiting GPI - in particular differentiating it from alcoholic insanity and cerebral syphilis: W J Mickle, 'Discussion on General Paralysis: The Delimitation of General Paralysis of the Insane', BMJ, Sept 29, 1906, pp 741 - 744.

suggestion is not taken for granted, but is based upon the observation that a core cluster of physical and mental signs remained recognisable as central to GPI throughout its history. These consisted of some form of progressive insanity, disarticulated or trembling speech, muscular weakness, and striking physical decline usually ending in death. A number of other signs - such as incoordination and loss of reactivity of the pupils - surrounded these, but the diagnosis was made quite readily without them. By the mid-nineteenth century these core features had given GPI a strong and coherent identity in the eyes of British doctors which would survive both peripheral confusion about the definition of the disease and important changes in imagery. I shall argue, furthermore, that even such important shifts in understanding as I have outlined above - including the new diagnostic opportunities which the Wassermann test brought - were probably more ideological in their impact than practical, and affected only the penumbra of GPI diagnoses. Documented epidemiological patterns of GPI which I shall discuss - although rather inadequate means of interpretation - bear out this supposition.

In suggesting that it is meaningful to follow GPI as a relatively stable entity, I am not entering into speculation about its underlying organic 'reality'. Neither is it my aim to undertake detailed retrospective studies of how GPI was classified, or to claim a standard of accuracy against which we might measure the diagnostic efforts of earlier practitioners. I do, however, suggest that whilst an inter-War doctor would have interpreted the disease - and particularly its cause - differently from a mid-Victorian doctor, the two would have agreed upon the majority of those patients who should receive the diagnosis of 'GPI'. Moreover, this relatively stable perception

allowed successive generations of doctors to use GPI, rhetorically, as a paradigmatic disease.

The professional history of psychiatry is the most important context in which my own history of GPI is written. The disorder came to prominence in Britain around 1840, just as the establishment of the Central Lunacy Commission (under the auspices of the Lunatics Act, 1845) paved the way for the first nation-wide organisation of asylums. Over the following century the fate of GPI would in some ways mirror the fate of the asylums. Until 1900 it would represent an ever increasing proportion of asylum inpatients, and would reflect in its degrading course the expansion and progressive demoralisation of the asylum system. During the twentieth century it would gradually decline and, with the advent of malaria therapy, more and more of its victims would find themselves ending their days outside the asylum walls - just as psychiatry as a whole attempted to change its emphasis from institutional to community care. Against this setting, alienists on the one hand watched the progress of the disease with a sense of powerlessness; on the other hand found that it could be used to make important claims for their profession. Much of the history of psychiatry has focused upon the attempts of practitioners to raise their scientific or medical status through a variety of strategies.[24] As many of the rather cursory secondary accounts suggest, this theme was indeed an extremely important aspect of GPI's

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[24] See for example A Scull, Social Disorder/Mental Disorder (London: Routledge, 1989); Idem, The Most Solitary of Afflictions: Madness and Society in Britain 1700 - 1900 (New Haven and London: Yale University Press, 1993); Idem, 'Somatic Treatments and the Historiography of Psychiatry', Hist Psychiatry, 1994, 5, pp 1 - 12; W F Bynum, 'Theory and Practice in British Psychiatry from J C Prichard (1786 - 1848) - Henry Maudsley (1835 - 1918)', in T Ogawa (ed), History of Psychiatry: Mental Illness and its Treatments (Tokyo, Japan: Saikon, 1982), pp 196 - 216; L S Jacyna, 'Somatic Theories of the Mind and the Interests of Medicine in Britain, 1850 - 1879', Med Hist, 1982, 26, pp

history - and it emerges in successive chapters of my thesis. Most obviously, it informed debates about the very identity of the disease during the mid-nineteenth century, as alienists attempted to establish their status with respect to adjoining specialties and professions. Here the concerns of neurologists and alienists to a certain extent impinged upon each other, since the history of tabes dorsalis - a progressive paralysis not linked to insanity - often ran in parallel to that of GPI. Tabes itself has received little attention from historians - although its identity as a physical disorder with no prominent mental elements partly explains this.[25] Its association with syphilis foreshadowed that of GPI by ten to twenty years; and at the beginning of the twentieth century both diseases were united under the new category of 'neurosyphilis'. Despite the intellectual relationship between the two diseases, however, the fates of each type of patient were very different; and the reasons for this throw light upon the differences between the nineteenth-century psychiatry profession, and the neurology profession which began to take shape through a number of specialist hospitals from the 1860's onwards. Although tabes and neurology are not the major story in my thesis, they recur at intervals throughout it as they demonstrate how psychiatrists viewed their relationship to neighbouring specialties.

During the twentieth century, the application first of laboratory techniques, then of malaria therapy, provided new ways of using GPI to make claims for the

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233 - 258; I R Dowbiggin, Inheriting Madness: Professionalization and Psychiatric Knowledge in Nineteenth-Century France (Berkeley & Los Angeles, Ca: University of California Press, 1991).

[25] Apart from traditional accounts in, for example, L C McHenry (ed), Garrison's History of Neurology (Springfield, Illinois: Charles C Thomas, 1969), p 425 et seq; and J D Spillane, The Doctrine of the Nerves: Chapters in the History of Neurology (Oxford: Oxford University Press, 1981), the fullest account deals only with the early history of the disorder: F Schiller, 'Venery, the Spinal Cord, and Tabes Dorsalis before Romberg', J Ment Nerv Dis, 1976, 163, pp 1 - 9.

status of psychiatry. But claims for the disease - although an important part of GPI's history - tell only one story. Modern scholarship, focusing particularly upon the advent of the laboratory, has attempted to show how science is often used more for its rhetorical than for its practical benefits.[26] In the general history of psychiatry too this is a common theme; and predictably it is a crucial aspect of GPI's 'scientific' identity. I have therefore tried where possible to compare rhetoric - as expounded in journals and books - with practice as indicated by other forms of evidence such as asylum records. This approach demonstrates time and again how often there was a gulf between the claims of psychiatrists for their flagship disease, and the more tangible results that they aspired to.

The histories of GPI and syphilis converged towards the end of the nineteenth century, as first continental, and subsequently British doctors accepted that the venereal taint was the prime cause of GPI. Syphilis - far more than GPI - has attracted quite extensive historical interest as a multi-faceted disease which throws light upon sexual morality, gender relationships, and approaches to disease control and prevention. An earlier generation of accounts traditionally traced the clinical elucidation of the disease as a heroic medical enterprise, concentrating in particular upon key strides forward: the separation of syphilis by Ricord from a vague conflation of venereal disorders; the accumulation of clinical and pathological evidence to elucidate the remote, 'tertiary' effects of the disease; the development of the diagnostic Wassermann test during the early twentieth century; and the (not quite)

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[26] See for example S E D Shortt, 'Physicians, Science and Status: Issues in the Professionalization of Anglo-American Medicine in the Nineteenth Century', Med Hist, 1983, 27, pp 51 - 68.

final defeat of the disease through drug treatment and public health campaigns.[27] Quetel and Oriel provide probably the most comprehensive chronological accounts which - although retaining elements of medical triumphalism - also offer rich social and professional detail, and are the most obvious starting-points for understanding the various ramifications of the disease.[28] Nevertheless whilst GPI naturally has a place in such histories as a successful piece of elucidation, it generally only appears in passing.[29] In fact the forging of the link between GPI and venereal disease was far from being a self-evident piece of science, but was the result of basic changes in the conception of syphilis, and of alterations in what doctors considered to be acceptable criteria for causal association. These 'medical' conditions for making the link were, furthermore, closely tied to broader cultural changes in the perception of venereal disease towards the end of the nineteenth century.

The social, sexual and moral aspects of syphilis have attracted far more interest than the medical aspects.[30] The first key historiographical tradition for the nineteenth century concerns prostitution, and in particular the socio-political debates surrounding the Contagious Diseases Acts in Britain.[31] Although such accounts -

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[27] See, for example, C C Dennie, A History of Syphilis (Springfield, Illinois: Charles C Thomas, 1962).

[28] C Quetel, History of Syphilis (Oxford: Polity Press, 1990); J D Oriel, The Scars of Venus: A History of Venereology (London: Springer-Verlag, 1994). See also J Cleugh, Secret Enemy: The Story of a Disease (London: Thames and Hudson, 1954).

[29] Rosen views the link with syphilis retrospectively, asking why it was made so late. He concludes that the answer lies in doctors' adherence to non-specific theories such as degeneration, and in the prior need to clarify GPI's clinical identity: Rosen, 1968.

[30] See A M Brandt, No Magic Bullet: A Social History of Venereal Disease in the United States Since 1880 (New York, Oxford: Oxford University Press, 1987); J Cassel, The Secret Plague: Venereal Disease in Canada 1838 - 1939 (Toronto, Buffalo, London: University of Toronto Press, 1987); R P T Davenport-Hines, Sex, Death and Punishment (London: Fontana, 1991); R Pearsall, The Worm in the Bud: The World of Victorian Sexuality (London: Pimlico, 1993).

[31] J R Walkowitz, Prostitution and Victorian Society: Women, Class, and the State (Cambridge, New York: Cambridge University Press, 1980). For an account of similar issues in French history, see J

which necessarily focus upon gender politics - deal with a period prior to the linkage of GPI and syphilis, they provide the context for understanding how the spectre of syphilis as an insidious social evil grew in the public and medical minds. This perception would pave the way for imputing progressively more, seemingly unrelated, medical disorders to venereal disease, and for accepting to a certain extent the congenital implications of syphilis - if not the broad sweep of evils suggested by Fournier. The second key historiographical tradition - and one which provides rich material for analysis - surrounds the incorporation of syphilis into debates about degeneration. The history of degeneration itself has an exhaustive secondary literature; and its intersection with the history of syphilis has received less, but increasing, attention. The French historian Corbin describes the flowering of the concept of congenital syphilis in France, and describes how it came to epitomise the fear of unbridled sexuality at the turn of the century. He focuses in particular upon the use of this concept in aiding Fournier's imperialist project to expand the venereology profession in France.[32] In the British context, there have been similar accounts of the idea of congenital syphilis - although a surprising silence upon the concomitant development of a coherent venereology specialty.[33] A handful of these accounts turn to the implications of the link between syphilis and GPI at the turn of the century. Thompson, in her study of Clouston and the Morningside asylum, places

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Harsin, Policing Prostitution in Nineteenth-Century Paris (Princeton: Princeton University Press, 1985).

[32] A Corbin, 'Hereditary Syphilis or the Impossible Redemption: A Contribution to the History of Morbid Heredity' in Idem, Time, Desire and Horror: Towards a History of the Senses, transl J Birrell (Oxford: Polity Press, 1995).

[33] See, for example, E Lomax, 'Infantile Syphilis as an Example of Nineteenth-Century Belief in the Inheritance of Acquired Characteristics', J Hist Med, 1979, 34, pp 23 - 39.

the general paralytic in the context of the moral ambivalence of Scottish alienists, and suggests an increasing repugnance towards the general paralytic after the link with syphilis was made.[34] Showalter, in her discussion of literary mythologies of the fin-de-siécle, shows how the imageries of congenital disease, sexual perversity, and violence coalesced in the figure of the male general paralytic, who became the archetypal 'villain' of the late nineteenth century: 'the reprobate whose decline into vice paralleled the decline of the prostitute.'[35] The cross-fertilisation of negative imagery between the two diseases seems obvious; and I would argue that it played an important part in their medical linkage. However, I aim to balance such assumptions by drawing out an alternative history of the general paralytic: one in which the potential patient was seen as the epitome of admirable and vigorous - if uncontrolled - manhood. This depiction, drawn from epidemiological, clinical, and social conceptions, remained strong despite increasingly degenerative connotations from the turn of the century onwards.

A third aspect of syphilis which has attracted historical attention is the acceleration of public health interventions which began during the second decade of the twentieth century, spawning debates upon the moral and clinical merits of

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[34] M S Thompson, 'The Wages of Sin: The problem of alcoholism and general paralysis in nineteenth-century Edinburgh', in W F Bynum, R Porter, and M Shepherd (eds), The Anatomy of Madness: Essays in the History of Psychiatry (London and New York: Tavistock Publications, 1985 - 1988), 3 Vols. Vol 3, pp 316 - 340.

[35] E Showalter, 'Syphilis, Sexuality, and the Fin-de-Siècle', in R Yeazell and N Hertz (eds), Sex, Politics, and Science in the Nineteenth-Century Novel: Essays from the English Institute (Baltimore: Johns Hopkins University Press, 1986), pp 88 - 115; Idem, The Female Malady: Women, Madness and English Culture, 1830 - 1980 (New York: Penguin, 1985), p 111. Spongberg also develops these ideas, see M Spongberg, Feminising Venereal Disease: The Body of the Prostitute in Nineteenth-Century Medical Discourse (forthcoming, 1998), esp Chs 8 and 9.

prophylactic and educational measures.[36] This development was closely associated with the organisation of the discipline of venereology - particularly through the establishment of Government-funded venereal disease clinics - although again this potentially rich subject of specialisation has received surprisingly little attention in the British context. The period is the backdrop to key changes in the medical management of syphilis through the application of laboratory techniques - particularly the diagnostic Wassermann test. GPI entered into these advances as psychiatrists applied the test widely to general paralytics, both to elucidate their syphilitic histories, and to identify and monitor their disease. Whilst the advent of the laboratory - and specifically its relationship to clinical forms of knowledge - has attracted much historical attention, its application to psychiatry has attracted far less. Of greatest interest for my purposes is an account by Ludwig Fleck of the development of the Wassermann test in the context of an early sociology of scientific knowledge.[37] Although Fleck deals with general syphilis rather than psychiatry itself, he provides an excellent starting-point for understanding the ambivalent attitudes of contemporary clinicians, including psychiatrists, towards using this laboratory tool in their practice. Echoing the recurrent theme of my thesis, this episode illustrates both strenuous attempts to use GPI's new laboratory status to make claims for psychiatry; but also the arguable gulf between rhetoric and practical gains.

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[36] See, for example, M W Adler, 'The Development of the Venereal Disease Services', in S Farrow (ed), The Public Health Challenge (London: Hutchinson, 1987), pp 107 - 111; D Evans, 'Tackling the 'Hideous Scourge': The Creation of the Venereal Disease Treatment Centres In Early Twentieth Century Britain', Soc Hist Med, 1992, 5, pp 413 - 433; S M Tomkins, 'Palmitate or Permanganate: The Venereal Prophylaxis Debate in Britain, 1916 - 1926', Med Hist, 1993, 37, pp 382 - 398.

[37] L Fleck, Genesis and Development of a Scientific Fact, transl F Bradley and T J Trenn (Chicago, London: University of Chicago Press, 1935; reprinted 1979).

I conclude with two provisos. First, the main focus of this thesis is the response to GPI in Britain. Nevertheless France seemed to the British - particularly during the nineteenth century - the cradle and headquarters of the disease, and the work of French doctors such as Bayle and Fournier was vital and far-reaching. I therefore spend some time considering French work specifically as it affected the ideas of British psychiatrists. Other countries which I mention only in passing deserve their own detailed research. In Germany, for example, there was a stronger tradition of neuropsychiatry which may well have given rise to different debates about the organic nature of GPI and its relationship with tabes - particularly as here GPI provided an overt model for classifiers such as Kahlbaum and Kraepelin. Scandinavian history remains shadowy to British historians, but here the unique organisation of hospitals and asylums played a part in encouraging doctors to accept the link between GPI and syphilis twenty to thirty years before it was accepted in other parts of Europe. In America, stronger notions of capitalist ambition and neurasthenia gave peculiar resonances to the disease. Here also funding for large-scale laboratories allowed syphilitic spirochaetes to be identified in the brains of general paralytics for the first time - a discovery which was full of nationalistic importance for American commentators. Developments in each of these countries would provide interesting comparisons to the situation in Britain but are, sadly, beyond the scope of my work.

Second, my thesis is primarily a history which bears upon the views and interests of doctors. Published accounts in journals are my main source of information, together with some archival material from asylums - although this is less

extensive. The absence of a coherent history from the point of view of patients is partly one of chosen emphasis, and partly due to the dearth of primary material which expresses the patient's voice. This situation changed to some extent with the advent of malaria therapy, when patients - now treated at an earlier stage in their disease - emerged from the shadows for the first time. Their life histories were documented in greater detail, they appeared to have an increasing degree of choice concerning their treatment, and their hopes and disappointments were increasingly expressed. Despite these changes - which had much to do with the general move towards voluntary treatment in psychiatry - the general paralytic and his family remain figures whose distress is understood only at second hand. But it is the sad image of the patient - an image which surely bears a considerable relation to reality as well as to the expectations of doctors - which endures strikingly throughout the history of GPI. The following passage, written by a mid-nineteenth-century alienist, expresses the tragic interest elicited by the plight of the general paralytic, which would last despite the changes in his fortune and in the fortunes of those caring for him:

'I conclude with a single reflection. Medical writers and poets have vied with each other in attempts to depict in glowing colours the painful interest attending the gradual but certain progress of decay in patients labouring under phthisis. It appears to me that the most graphic description of this disease would fade, when compared with a faithful portrait of General Paralysis. This disease suddenly seizes its victim in the prime and vigour of life, when he has acquired, by unexampled industry it may be, a high position in the profession which he adorns ... Arrested suddenly in the height of prosperity [he] gradually degenerates into a state of hopeless fatuity, and dies when

far beyond the reach of friendly consolation ... It adds, I think, painfully to the features of the disease, to see so much boasted grandeur and wealth associated with so much physical and mental degradation.'[38]

[38] D Skae, 'Contributions to the Natural History of General Paralysis', Ed Med J, 1859-1860, 5, pp 885 - 905; on p 905.

CHAPTER 1

THE MAKING OF 'GENERAL PARALYSIS OF THE INSANE'

Introduction

'When the great French physicians ... first seized on a particular group of mental symptoms ... found they had a regular sequence and history, that they were connected with certain obvious departures from the normal state of the brain ... and when they gave their generalisation a name and called it 'General Paralysis of the Insane', they sounded the death-knell of all the metaphysical and spiritual theories of insanity ... Its importance to the study of insanity was incalculable. I believe that the further and fuller study of general paralysis will do more for the elucidation of many of the unsolved problems as to the connection of mind with brain ... than that of any other disease.'[1]

With this rather biblical pronouncement, the Scottish alienist Thomas Clouston expressed an enduring belief in the importance of GPI to his profession - and one which has lasted to the present day. Histories of psychiatry routinely stress the precariousness of a specialty 'always ... but a step away from a profound crisis of legitimacy'[2]. Nineteenth-century practitioners made continuous attempts to achieve mainstream medical credibility by adopting somatic approaches to mental disease - from the enthusiastic uptake of phrenology and Parisian pathological anatomy during

the first half of the century to the later adoption of cell theory, neurophysiology, and degenerationism.[3] GPI has received relatively little attention in the context of this broader history of psychiatry, but the few accounts available usually take for granted one theme: its perfect representation of the medical model for those who studied and cared for its victims.[4] 'Medical' in this context refers to several characteristics: its association with observable brain lesions; its relatively predictable clinical natural history; its juxtaposition of physical with mental symptoms; and - latterly - its syphilitic aetiology. As a medical model par excellence, GPI has been serviceable to historians with various ideological viewpoints. Hunter and Macalpine, in line with their triumphalist approach and stringent physicalism, regarded the discovery of GPI as 'the climax of the age-old quest for a pathology of insanity ... the apotheosis of mental illness which shaped the course of psychiatric thinking ...'[5] Thomas Szasz, challenging the medicalisation of psychiatry, regretted that the specialty should have seized upon such an inappropriate role-model: '... Hence the ceaseless manufacture of disease names in psychiatry, together with a total lack of evidence that any of them ... are caused by demonstrable brain lesions on the model of paresis. It is the greatest scientific scandal of our scientific age.'[6]

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[1] T S Clouston, 'The Morisonian Lectures on Insanity for 1873 by the late D Skae: Lecture 6 (General Paralysis)', J Ment Sci, 1875, 21, pp 189 - 204; on p 189.
[2] A Scull, 1989, p 21.
[3] See, for example, Scull, 1989, esp Ch 6; Idem, 1993; Idem, 1994; Bynum, 1982; Jacyna, 1982; Dowbiggin, 1991; T M Brown, 'Mental Diseases', in W F Bynum and R Porter (eds), Companion Encyclopedia of the History of Medicine (New York: Routledge, 1993), 2 Vols. Vol I, pp 438 - 436.
[4] For a straightforward short account, see Zilboorg and Henry, 1941, pp 526 - 551.
[5] Hunter and Macalpine, 1963, p 780. In a similar vein, Edward Hare described the 'efflorescence of genius which led to and was evoked by the discovery of dementia paralytica ... one of the most astonishing and glorious chapters in psychiatry ...', Hare, 1959, pp 605 - 6.
[6] Szasz' main target in this article was the use of GPI's syphilitic aetiology as a model for medicalising all mental disease: Szasz, 1976, p 315. Compare Ackerknecht, who apologetically noted that the high

But did GPI really make an impact upon nineteenth-psychiatry - for good or for ill - as such impassioned accounts suggest? More recently Berrios has challenged this view: 'In general, there is little evidence that alienists considered General Paralysis as a "paradigm disease", ie as a model for all other mental diseases. It can even be said that the new 'disease' created more problems than it solved'.[7] He denies, first, that nineteenth-century alienists were rigidly organic in their approach and, second, that the concept of GPI as an agreed disease entity was ever stable enough to be used as a suitable model. The traditional account is, he suggests, 'a useful myth [which] has been kept alive by twentieth-century historians who wish to write off all psychiatry before Freud as prehistorical organicism'.[8] Certainly the traditional stories are simplistic, and often transparently informed by political or ideological viewpoints. However, I refute Berrios' claim that alienists did not consider GPI a paradigmatic disease. On the contrary, I suggest that they constantly and emphatically used it as a model to express their hopes for a more rigorous and persuasive psychiatry.[9] Organicism, pace Berrios, was indeed an ideal which much of the rhetoric surrounding the disease expressed. In addition, as I have argued, GPI was certainly regarded as conceptually stable enough to be set up as such a model.

A series of themes, considered in this chapter, demonstrate how alienists sought to present GPI as a flagship for their profession. First, from the 1840's to the

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proportion of general paralytics in asylums 'excuses the dominance of brain psychiatry of the latter nineteenth century', Ackerknecht, 1959, p 66.

[7] Berrios and Porter, 1995, p 39.

[8] Berrios, 1985, p 394.

[9] R Smith makes this point in passing: 'A modern view that GPI provided a model for understanding insanity (by standardising the natural history of a disease and then linking it to physical disorder) must be applied with caution to the nineteenth century. But there is no doubt that physicians believed in an

1860's, they struggled to formulate GPI as a natural disease entity, and to understand upon what basis this disease status could be claimed. In particular they grappled with the question of how insanity was related to the physical symptoms of GPI; and for the majority, a physicalist stance led naturally to an insistence that both insanity and paralysis were integral to the disease - in fact, part of the same disease process. This question was of great importance when alienists studied the very early stages of GPI, and many explicitly linked it to their own claims for expertise and responsibility over the general physician. Organicism, specialty status, and the very definition of GPI were thus clearly interlinked. During the 1860's and 1870's, when the disease status of GPI seemed assured, it was used far more overtly as a symbol of the promise of organicism and 'natural' symptomatology - themes which were indeed preoccupations for most alienists, even if they were quite aware of being unable to reach these ideals in practice. Many times during the latter half of the century claims for the disease reflected concerns about psychiatry's relationship to the medical profession at large, to education, to the law, and to the emerging speciality of neurology. Simultaneously, alienists began to tell the triumphant story of its discovery and description - so that the history of the disease (although largely played out in France) became part of its perceived 'flagship' role for the future of psychiatry. Claims for the importance of GPI were commonplace amongst alienists despite some fluidity in its clinical parameters; certainly until the 1890's (and probably beyond) they saw these as largely irrelevant compared with the convincing unity of the disease. Throughout this thesis I

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abstract model of progress along these lines ...', R Smith, Trial by Medicine (Edinburgh: Edinburgh University Press, 1981), p 35.

am concerned to show, as far as possible, how rhetoric differed from practice; how, for example, asylum conceptions of the status of GPI gave a different picture from published writings. This gulf is particularly striking when we consider the flagship role of GPI. The claims and arguments made for the disease were largely aired in journals, association discussions, and text-books; and it is clear (as Berrios would surely agree) that GPI's practical or theoretical impact upon the profession fell far short of alienists' hopes. This, of course, was a story repeated throughout the history of the specialty; but it does not mean that the symbolic importance accorded to GPI can be underestimated.

The Arrival of GPI

During the 1820's, Parisian alienists began to document a form of generalised paralysis peculiar to a proportion of the insane. The paralysis consisted of relatively constant and progressive physical features: disturbance in articulation, typically causing tremulous and indistinct speech; unsteadiness of gait leading to staggering; and eventually complete paralysis of voluntary movements, inability to swallow, incontinence, gangrene, and an undignified death. 'General paralysis' was soon well-recognised amongst the inmates of asylums; but an immediate problem concerned the relationship between this physical disorder and the mental symptoms which always seemed to accompany it. Early commentators such as Esquirol and Delaye were in no doubt that it was a severe complication of any form of insanity, much on a par with

epilepsy and apoplexy.[10] However the precocious young alienist Antoine-Laurent Bayle, pupil of the Charenton physician Royer-Collard, challenged this assumption in two pamphlets published in 1822 and 1826. The disorder, he suggested, was not a mere complication of insanity, but an integral part of a single disease made up of a triad of phenomena: madness characterised by ambitious monomania; progressive generalised paralysis; and the pathological lesion of chronic arachnitis.[11]

According to traditional accounts, Bayle simply discovered an objective disease entity - and the more detailed elucidation of GPI naturally followed. In reality, alienists were faced with a set of associated phenomena - insanity, paralysis, and brain lesions - and no self-evident ways of interpreting these phenomena. In the face of this fluidity, context and expectations were often important. Bayle, committed to the ideology of the emerging Parisian pathological anatomy school, based his claim for disease unity primarily upon what he perceived as a constant and specific pathological lesion.[12] In contrast Georget, in his 1826 critique of Bayle, articulated research principles drawing strongly upon Pinel's tenets. The disease, he suggested, should be defined by consistently linking a set of clinical phenomena - a specific form of insanity and progressive general paralysis - and not by working backwards from a

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[10] J -E -D Esquirol, Article on 'Dementia' in Dictionnaire des Sciences Médicales, Vol 7 (Paris: C L F Panckoucke, 1814), pp 283 - 300; J-B Delaye, Considerations sur une éspece de paralysie, qui affecte particulierement les aliénés (Paris: Masson, 1824).

[11] A-L-J Bayle, Récherches sur l'arachnite chronique, la gastrite et la gastro-enterite chroniques, et la goutte, considérée comme causes de l'aliénation mentale (Paris: Didot le Jeune, 1822), transl in M Moore and H C Solomon, 'Contributions of Haslam, Bayle, and Esmarch and Jessen to the History of Neurosyphilis', Archives of Neurology and Psychiatry, 1934, 82, pp 807 - 829; A-L-J Bayle, Traité des Maladies du Cerveau et de ses Membranes (Paris: Gabon, 1826).

[12] His 1822 paper made this statement only with respect to chronic arachnitis; in 1826, however, he broadened his claims by suggesting that the majority of mental disease was directly associated with a pathological lesion, and that chronic arachnitis simply provided a clear example of this doctrine.

putative pathological lesion.[13] Esquirol persisted in regarding general paralysis as a complication rather than a discrete disease entity, and his stance can be attributed to a number of factors: the variety of forms of insanity associated with paralysis; its apparently late onset in the chronically insane; an antipathy to organicism conditioned by his adherence to moral therapy; an aversion to the reduction of his own category 'ambitious monomania' to a mere symptom of chronic arachnitis; and a desire to broaden alienists' claims to prognostic expertise.[14] On a more mundane level, there were vast disagreements about Bayle's pathological findings, and a general denial of his rigid progression of mental symptoms. The young alienist was widely regarded as over-confident in his theory, to the extent of overlooking observational discrepancies; his contemporary Calmeil - who was, in contrast, overtly cautious in his statements - was far more warmly received. Although Calmeil clearly treated 'paralysie générale des aliénes' as a species apart, and tentatively attributed both mental and physical symptoms to a constant lesion ('encephalite chronique'), he fought shy of claiming that the lesion defined the disease. Neither did he claim that any specific form of

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[13] E-J Georget, 'Traité des maladies du cerveau et de ses membranes, par L J [sic] Bayle', Archives Générales de Médecine, 1826, 12, p 323: quoted in Brown, 1994, p 249.

[14] J-E-D Esquirol, Des maladies mentales considérées sous les rapports médical, hygiénique et médico-légal (Paris: J-B Baillière, 1838); transl E K Hunt as Mental Maladies: A Treatise on Insanity (Philadelphia: Lea & Blanchard, 1845), p 439. Bayle's fate was closely linked to these theoretical antagonisms. When his mentor Royer-Collard - a rival of Esquirol - died, Bayle was ousted by the Esquirol circle from his position at the Charenton, and was forced to take up a librarianship at the Paris Medical Faculty. The above account serves only as an introduction to the English debates; for a fuller consideration of the French arguments, see J D Hurn, Some Historical Aspects of General Paralysis of the Insane, 1820 - 1860 (unpublished MSc dissertation, 1994); Brown, 1994.

madness was observed; Bayle's ambitious monomania was, he suggested, a striking feature present in many, but not all cases.[15]

In France, then, many alienists were reluctant to create a new disease entity, and the observation of physical paralysis remained the only unshifting point of reference in a sea of often contradictory observations. British doctors would inherit these problems of disease definition as they became increasingly familiar with the new disorder. The alienist George Man Burrows (founder of the private hospital The Retreat) visited several Parisian asylums in 1817 and 1822, and a few years later described Bayle's work in his textbook of mental medicine - one of the first clear references in a British publication. Paralyses of all kinds, he noted, were common complications of insanity; Bayle, however, had described a 'peculiar species' associated with chronic meningitis, hesitation of the speech and partial weakness of the lower limbs. It was this last physical feature which Burrows selected as the basis for his English name: 'Incomplete Paralysis'.[16] Over the following years, British alienists began to describe the disorder in increasing numbers - and with growing conviction. Burrow's original term was quickly discarded, and by the 1830's the name 'general paralysis' or 'general paralysis of the insane' was commonly understood.

Its florescence in the asylums of Paris seemed puzzling to doctors, as did its apparent novelty, and comparative infrequency in Britain. Until mid-century it was

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[15] L Calmeil, De la Paralysie, Considérée Chez les Aliénés (Paris: Bailliére, 1826); Calmeil described the clinical features as: mental enfeeblement, disarticulation of the tongue and incoherent speech, and a disorder of mobility. Esquirol actively supported Calmeil at the expense of Bayle.

[16] G M Burrows, Commentaries on the Causes, Forms, Symptoms and Treatment Moral and Medical of Insanity (London: T&G Underwood, 1828); reprinted in Hunter and Macalpine, 1963, pp 777 - 783.

regarded as a primarily French disease; and in keeping with nationalist arguments about insanity as a whole, this identification was rarely flattering - whether it reflected character, society, or medical organisation in France. Burrows, for example, initiated a mildly acrimonious exchange with Esquirol when he suggested that poor asylum conditions might partly explain the far higher numbers seen in France.[17] On the contrary, Esquirol insisted, patients were paralytic well before admission: 'I am convinced', he added, 'that when [British alienists] have learned better to distinguish the symptoms of paralysis which complicate insanity, they will find ... particularly at London, as many insane paralytics as at Paris.'[18] The majority of British alienists were indeed willing to admit that they had to 'learn to see'; and that the unfamiliar disease had probably been under-recognised in their own country. John Conolly, champion of non-restraint at Hanwell Asylum, recounted his own initiation into GPI: he admitted that he had never noticed a case when he first read Calmeil's description in 1826; but having been shown several patients during a visit to the Charenton, he began to increasingly notice the disease at home.[19] Careless classification in public asylums was commonly blamed for this lack of recognition, as well as the common refusal of private asylum keepers to admit paralysed patients whom they regarded as incurable.[20]

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[17] Burrows 1828. Burrows estimated that French figures were ten times those of Britain.
[18] Esquirol, 1838 (transl 1845), p 439.
[19] J Conolly, 'Clinical Lectures on the Principal Forms of Insanity: Lecture XI', Lancet, Feb 28, 1846, pp 233- 237; on p 233.
[20] Prichard, for example, complained: 'Patients are dismissed from Bethlem when they manifest any indication of paralysis, and the events of such cases cannot ... be correctly noted.' J C Prichard, A Treatise on Insanity (London: Sherwood, Gilbert & Piper, 1835), p 109. This situation would continue throughout much of the century for private hospitals: see the comments of H Tuke, 'On General Paralysis', J Ment Sci, 1858, 5, pp 575 - 584; on p 581. I further consider views about the antiquity of GPI in Chapter 5 below.

During these early years, GPI was put to use for the first time in British alienists' pleas for the status of their specialty. Despite its growing appearance amongst the insane the majority of the medical profession were, alienists claimed, still almost completely ignorant of the disease. This observation quickly came to symbolise the isolation of mental medicine from mainstream practice. One of John Conolly's Croonian lectures of 1849 was devoted to GPI which, although now common in asylums, was 'to many experienced medical men entirely unknown.'[21] Prominent alienists would echo this complaint for at least ten years, arguing that general ignorance of an important new disease demonstrated the low esteem in which they were held. Ten years after Conolly, Harrington Tuke noted how common it was for ordinary medical attendants to dismiss the need for consultation with an alienist, and to diagnose the rest cure for 'nervous strain' which quickly proved to be GPI. Amongst ordinary physicians, he complained, GPI was 'almost unknown ... Thus ... general medicine ignores the branch of alienist physicians, disdaining them as having the taint of trade ... I have in no way over-estimated the importance of this disease and the ignorance of the profession at large as to even its existence...'[22]

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[21] J Conolly, 'The Croonian Lectures: Lecture III: Description of General Paresis', Lancet, Oct 27, 1849, pp 443 - 446; on p 443.

[22] H Tuke, 1858, pp 575, 580-1. See David Skae's comment that GPI was increasing in frequency yet under-recognised: 'I have met with very few practitioners who had a correct idea of this disease ...': Skae, 1859 - 1860, p 885. See also Austin, 1857, extract in Hunter and Macalpine, 1963, p 1054.

Creating 'GPI'

GPI was clearly already close to the hearts of alienists; but these complaints came as they were still struggling to establish its very identity. British authors such as Burrows and Conolly tended to regard GPI as a 'calamitous' physical complication of insanity until the 1840's; partly because the non-specific mental symptoms shifted attention to the well-defined and specific physical features.[23] Over the next twenty years, however, GPI would become a disease in its own right. The basis upon which this claim could be made had been suggested in the French debates: specific and constant brain pathology; a predictable clinical natural history; and correlation between the two. The alienist Harrington Tuke wrote copiously about GPI from the 1850's, and demonstrated well the struggle to raise it to disease status. To Tuke, the obvious organic basis of the disorder was not sufficient for this claim. Certainly gross brain lesions were constantly present at post mortem; and certainly there was a consensus upon some broad features - for example inflammation of the membranes and softening and atrophy of the surface cortex. Nevertheless, changes were acknowledged to be neither specific nor constant - and certainly by no means as characteristic as Bayle had claimed. Bayle's efforts were respected but, as in France, his dogmatism was received with suspicion; and it was Calmeil's work which was generally quoted until the 1860's.[24] Tuke, despairing of finding morbid appearances

[23] Connolly 1846, p 235. Burrow's suggestion that poor asylum conditions in France might allow the insane to become paralytic reflected this assumption that GPI was a complication of any form of insanity: Burrows, 1828.

[24] See Burrows' criticism of Bayle: 'Unquestionably, pathology is indebted to anatomy's studies ... Yet it is impossible not to suspect that too much enthusiasm and aptitude for theory have influenced the pursuit, and that extraordinary facilities for post-mortem ... sometimes advance a specific theory rather than advance truth ... This ingenious physician has attempted to prove too much, and more than facts

characteristic of GPI, resorted to those strategies which were serviceable for the pathological project as a whole. Committed to a vascular interpretation of neural pathology, he predicted that microscopy and chemistry would reveal such a basis for GPI - and chided those who demanded to see such as yet imperceptible features: 'It is true we may not always be able to demonstrate [its organic nature] by the scalpel or the microscope, nor has chemistry helped us to a solution of the mystery, yet we cannot but feel, that with such symptoms, it is hardly possible that a peculiar organic disease is not present, although it may escape our imperfect means of investigation.'[25] Predictably, the application of microscopy from the 1850's onwards did not solve any problems. Two opposing interpretations of the findings were vigorously debated until the end of the century: one (associated mainly with the French) claiming that primary inflammation caused vascular and nutritional disturbances of the cortex; another (associated mainly with the Germans) claiming that blood vessel hypertrophy was the primary change, leading to ischaemia, inflammation, connective tissue overgrowth, and destruction of the nerve fibres.[26] As in other areas of pathological research, differences of opinion were commonly blamed upon 'seeing' erroneously because of theoretical preconceptions.[27]

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warrant.': Burrows 1828, p 782. See also Bucknill and Hack Tuke's remark that GPI was 'unknown until Calmeil's admirable work': J C Bucknill and D H Tuke, A Manual of Psychological Medicine (Philadelphia: Blanchard & Lea, 1858), p 328.

[25] H Tuke, 'On General Paralysis', J Ment Sci, 1859, 6, pp 79 - 93; 198 - 205; 420 - 438; on p 85.

[26] For a fuller traditional account of pathological research into GPI see Zilboorg and Henry, 1941, pp 541 - 542.

[27] For a good non-British example of this, see the account by the German psychiatrist Westphal: C Westphal, 'On the Present State of our Knowledge Regarding General Paralysis of the Insane', transl from German by J Rutherford, J Ment Sci, 1868, 14, pp 162 - 192; 506 - 522; esp pp 176 - 190. Westphal's arguments about 'seeing' are reminiscent of Parchappe's thirty years earlier, see Hurn, 1994, pp 19 - 20.

GPI retained its kudos as the mental disease in which pathology was '... more evident, more characteristic, and more general than in any other form of insanity.'[28] Nevertheless because of the frustration of the pathological project, alienists found it more fruitful to turn their attention to its clinical natural history.[29] Bayle's depiction of GPI had stressed a progressive and predictable unfolding of mental and physical features. Although successive writers quickly rejected his dogmatic delineation of stages, and his exclusive stress upon ambitious monomania, there was soon a strong consensus as to the essential core features of the disease. For most British alienists these were insanity of some form accompanied by inarticulate speech, tremor of the lips and tongue, progressive muscular paralysis, and invariable fatality. The mental symptoms attached to the disease were agreed to be variable: following Bayle's work French alienists, for example, identified three clinical forms of 'manic-ambitious', 'melancholic-hypochondriacal' and 'dementia'.[30] The relative frequency of such varied symptoms would be perceived to change over time; but, British alienists regarded some kind of expansive insanity (from mild euphoria or extravagance to florid delusions of grandeur) as classic of the disease throughout its history and, as I discuss below, it was this feature which determined most strongly the prevailing popular image of the GPI patient.[31] Nevertheless, around the apparent stability of the physical symptoms, arguments raged about the nature and constancy of the associated

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[28] W Griesinger, Mental Pathology and Therapeutics, 2e, transl C L Robertson and J Rutherford (London: The New Sydenham Society, 1867), p 434.

[29] As Tuke concluded in the face of pathological inconsistency: 'Claims for a nosological entity must therefore come from the progress of symptoms.': H Tuke, 'On General Paralysis', J Ment Sci, 1861, 7, pp 278 - 285; on p 283.

[30] See Berrios, 1985, p 397.

insanity. From the 1840's, several French alienists challenged the idea that madness was central to the definition of the disease at all. In 1846 Réquin suggested that outside the asylums - in general hospitals and in private practice - doctors might find cases of general paralysis without any mental symptoms. A year later J-G-F Baillarger agreed that paralysis itself was the only fundamental feature of GPI, and that insanity often did not accompany it.[32] During the 1850's Scipion Pinel suggested dividing the disease into 'simple' and 'complicated' cases, in which insanity was absent and present respectively; alienists, he suggested, were only aware of those patients with the latter form who had become inconvenient or dangerous to society.[33]

My research does not take me into these vigorous debates in French circles. However, in the response of British alienists such as Tuke it is clear how important it was felt to retain insanity as central to the definition of GPI. From a practical point of view, alienists were still primarily custodians of the overtly insane; this theoretical question of GPI's identity could therefore have been dismissed as less important than the question of whether a particular patient needed admission to an asylum. Réquin and Pinel acknowledged this, and in one sense Tuke himself agreed that the central problem was one of pragmatism: alienists should simply cordon for themselves the forms of general paralysis which obviously manifest with insanity. Thus, agreeing that a form of general paralysis might exist without mental symptoms, he noted:

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[31] Tuke, for example, noted that there was practically always an expansive edge to the insanity at some stage in GPI: Tuke, 1859, p 427.

[32] Baillarger's ideas were complex: he later seemed to contradict this view, claiming that he had always regarded dementia (rather than delirium) as fundamental to GPI. British alienists appeared to associate him with the first interpretation.

[33] The question was discussed at a meeting of the French Society of Medical Psychology in 1858: a tentative conclusion was reached that there may be forms without insanity, but that they were rare, and

'...[But] we must distinguish between 'simple' general paralysis, and our disease, which should be called ... Calmeil's 'paralysie générale des alienes'...'[34] This rather circular argument opened out, however, when alienists considered at what point they could confidently assert that mental symptoms were truly absent. Tuke's approach to this question during the late 1850's and early 60's clearly demonstrated his conviction that mental unsoundness must be assumed to accompany physical signs of GPI. He considered it 'unphilosophical' to leave insanity out of the fundamental definition of the disease, and denied that there was any period with physical manifestations alone, since one could never prove the absence of mental symptoms.[35] Terminology was important here, since the name 'general paralysis' had, he suggested, led to erroneous assumptions about the physical alone being fundamental.[36] Tuke's stance rested firmly upon his physicalist approach to mental disease: like the majority of British alienists he saw it as fundamental that mental processes had a physical basis. GPI displayed both physical and mental features in tandem: the unity of the disease thus affirmed that the same pathological process produced both - that they were inseparably connected: 'It is not doubted by anyone that [general paralysis] destroys the mind function just in the same way as it does the bodily one, through a physical

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were closely linked pathologically to those forms with insanity. Both Calmeil and Esquirol remained unclear as to whether GPI could exist without insanity. See Skae, 1859-1860.

[34] H Tuke, 1859, p 79.

[35] Tuke 1859, p 427.

[36] D Skae, 'Dr Harrington Tuke's Paper on the Diagnosis of General Paralysis', J Ment Sci, 1858, 5, pp 78 - 81; on p 78.

change in the nerve cells...'[37] Thus, wherever physical symptoms were manifest, mental symptoms must necessarily also be there - however hard they were to discern.

Apart from its philosophical basis, the greatest implication of this assumption for Tuke was the importance of discerning insanity which was not yet quite obvious in those patients who showed subtle early physical signs of paralysis. The ability to recognise these tell-tale physical signs - a tremulousnessness of the lips, stumbling over a syllable - was, Tuke argued, unique to the experienced alienist; and demonstrated his expertise compared with the ignorance of the general practitioner.[38] It was also an indefinable knowledge which could not easily be explained: 'The ear of the physician accustomed to the treatment of the insane, detects instantly the fatal lisp ... it is to him an unerring index ... of the stage which [GPI] has reached.'[39] The Edinburgh alienist David Skae attached a similar importance to early paralysis and to the 'GPI face': 'The so-called paralytic symptoms are the diagnostic, the significant, and the hopeless indications of this distressing malady ...'; '... A peculiar expression of the countenance, very difficult to describe, but so peculiar and so easy to recognise ... that anyone who has had a few years of experience among the insane could pronounce upon the existence of general paralysis from the aspect of the face alone.'[40] In both French and British writings, it was common to tell cautionary tales of patients who had been dismissed by ordinary attendants - perhaps casually prescribed a rest

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[37] Clouston, 1875, p 190. Griesinger's text made the same philosphical point: discussing the French theories of GPI without insanity, he urged that the disease must be defined as paralysis with insanity, because both arose from the same cerebral process: Griesinger, 1867, p 394.

[38] For a detailed account of the early speech defect see H Tuke, 1859, pp 198 - 205.

[39] H Tuke, 1859, p 200. Griesinger similarly stressed the sinister interpretation which the expert attached to a slight deterioration of speech: 'Wherever this is remarked ... [the patient] may with absolute certainty be considered as lost ...', Griesinger, 1867, p 398.

cure - only to be subsequently identified as doomed men by psychological physicians. Thus shocked, angry relatives would learn that their loved ones were victims of an insidious but fatal disease - one of which they had probably never heard before. Tuke quoted a story which Esquirol told of his unique ability:

'M., had become irritable, and easily excited at the slightest opposition, he had refused all medicine, asserting that he was never so well or so happy. Dr.- a physician, equally talented as esteemed, brought him to Paris, and to me. 'I commit to your treatment, (said he) a most interesting patient, who is suffering only from transient excitement, your care, and separation from scenes that appear to augment his disorder, will speedily restore him to health'. I converse with this patient, (continues Esquirol) he tells me of his projects for the future, of his present happiness, the acquaintances he and his family will gain by their visit to Paris, &c. After half an hour I am asked my opinion; it is, that the patient will not recover, that he is incurable, and that he has not one year to live. At the expiration of seven months, this gentleman sank under a malady, which, at it commencement, appeared so insignificant in its character even to so distinguished and practised a physician as Dr. K-.'[41]

Tuke's own similar story was typical:

'I met at a friend's house ... a clergy-man, aged fifty-four, a remarkably fine looking man; he had lived much in the country, and talked a good deal about horses and dogs, more than clergymen usually do ... after dinner he mentioned that he was

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[40] Skae, 1859 - 1860, pp 886, 887.
[41] H Tuke, 1859, p 81.

the best billiard player of his year at Cambridge, and the best Greek scholar; on some one noticing this, he added, with great eagerness, that he was the only one who understood the Digamma. I fancied there was some slight tremor about his lip ... I had never seen this gentleman before, but I was on intimate terms with our entertainer, and I mentioned to him my impression of his friend's condition, and my opinion that there were symptoms in him of a peculiar form of malady, which, if unchecked, would end in absolute insanity and paralysis. Eight months afterwards this gentleman was brought to my house, in which he died, as I anticipated he would do, with unequivocal symptoms of paralytic insanity.'[42]

Over the following years, alienists increasingly focused upon the mental 'prodrome' of GPI - small moral lapses or hints of forgetfulness which might be recognised by the skilful professional as the earliest signs of madness. A discussion between leading alienists during the following decade was typical. Here the general physician Gairdner discussed several cases of apparent general paralysis without insanity; the most that he could find was possible memory loss and mild emotional lability. In one patient, a personal friend of the doctor's, he noted '...certain misunderstandings which occurred towards the close of his useful and honoured life ... In no other respect ...could [he] have been pronounced technically insane...'[43] The gathered alienists who replied to his cases acknowledged the difficulties in assessing such disguised symptoms, but assured him that his patients <u>were</u> already mentally

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[42] H Tuke, 'On General Paralysis', <u>J Ment Sci</u>, 1860, pp 88 - 104; on p 98.
[43] W T Gairdner, 'Two Cases having Certain Points of Resemblance to General Paralysis of the Insane, but without Insanity; & Occasional Memoranda of a Third Case', <u>J Ment Sci</u>, 1876, 22, pp 249 - 261; on p 256.

unsound, and would become overtly insane in time. As the alienist Yellowlees ominously advised: 'Look out for that man's mind; I do think it will go...'[44]

This emphasis upon the alienist's competence to make an early diagnosis had, sadly, little to do with hopes for medical intervention. The diagnosis emphatically spelt doom; occasional claims that the course of the disease might be affected by doctors held no conviction, and there were no convincing reports of cure. To suggest that the rhetoric had more to do with the assumption of physicalism and claims of expertise is not to suggest that alienists such as Tuke were seeing insanity where it did not exist; this would be beyond the historian's ability to decide. One might place their claims in the context of a general project to increase the bounds of insanity during the 1860's and '70's - exemplified by Maudsley's campaign for his specialty's status. However, to consider an interest in equivocal signs of insanity as 'social pathology' would be stretching the point in the case of GPI. In the dinner-party example given above, Tuke clearly interpreted the clergyman's not-quite-acceptable conversation as early insanity - but he considered his view to be vindicated by subtle physical signs, and by the subsequent illness of the patient. He acknowledged, too, how difficult it was to distinguish between an extravagantly optimistic personality and mental unsoundness; and stressed that he did not suggest early confinement for the equivocally insane, but simply made allowance for any irregular acts - including criminal acts - which might follow.[45] Nevertheless, Tuke's view of GPI clearly symbolised strongly for him important ideologies in mental medicine. GPI as a mere

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[44] Gairdner 1876, p 335. Westphal also repudiated the idea of GPI without insanity; all showed, he claimed, 'slight, easily overlooked psychical weakness ... to which no importance is attached...', Westphal, 1868, p 521.

physical terminal event in any form of insanity said nothing about the nature of mental disease; GPI as an entity in which physical and mental were part of the same process made an important statement about the physicalist basis of insanity.

Whilst alienists continued to negotiate the exact mental and physical parameters of GPI, by the mid-to-late 1860's it was virtually unanimously accepted as a disease in its own right. Not until 1869 did the Medico-Psychological Association officially recognise this, as they endorsed a decision of the International Congress of Alienists that GPI was 'a distinct morbid entity, and not at all ... a complication, a termination of insanity.'[46] Text-books followed suit within a few years: Bucknill and Hack Tuke's manual, for example, listed GPI as a complication in 1862, but as a separate disease in 1874.[47] In asylums, the practice of assigning patients to categories in clinical records partially reflected this official consensus. At Hanwell, for example, the disorder was termed a physical complication of insanity until 1860. By 1870, it was clearly entered as a diagnosis in its own right. From 1880, however, following the introduction of a standard clinical form, the diagnosis was again practically always given as [insanity type] + general paralysis - a style which equivocally suggested a complication.[48] Such discrepancies perhaps illustrate the problematical relationship between psychiatric theory and the chaotic, pragmatic business of running an asylum - rather than suggesting serious contradictions. Whilst Berrios (focusing upon the

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[45] H Tuke, 1860, p 102.
[46] Report of a Quarterly Meeting of the MPA, 28 Oct 1869, J Ment Sci, 1870, 15, p 635.
[47] Bucknill and Tuke, 1862; 3e, 1874. In 1871, Blandford noted that GPI was now always classified as a separate disease: G F Blandford, Insanity and its Treatment (Edinburgh: Oliver and Boyd, 1871), p 287. See also G MacKenzie Bacon, 'General Paralysis of the Insane - its Nosological Position', J Ment Sci, 1871, 17, pp 206 - 210; on p 210: 'GPI is a special form of brain disease ... has a definite and

French situation) notes that disagreement continued as to its precise clinical boundaries, I would suggest that by the 1860's British alienists were confident that GPI was their most recognisable and robust disease entity, and one which they could use to great benefit in their professional arguments.[49]

GPI and the Insanity Defence

GPI, as representative of the physicalist ideology of insanity, was sometimes used in the long and acrimonious argument between British alienists and lawyers. The dispute, particularly intense during the 1860's and '70's, was regarded as exemplifying the low esteem in which the medical profession - and particularly asylum doctors - were held; lawyers, it was claimed, regarded alienists as benevolent apologists for criminal behaviour, believing that they would find signs of insanity 'wherever they looked earnestly for them'.[50] The intricacies of this debate are well-documented; Roger Smith in particular has demonstrated the near-impossibility of resolving differences between the legal and medical discourses of responsibility - which alienists portrayed as a battle between narrow legal precedent and medical truth gleaned from experience of nature.[51] In particular it seems that alienists, by

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regular course, with peculiar mental symptoms, which almost suffice to distinguish it from other forms of disease in which body and mind decay together ...'

[48] Clinical case-books of Hanwell Asylum (1850 to 1900). GLRO, H11/HLL/B19, 20.

[49] Berrios, 1995, p 39.

[50] H Maudsley, 'Stealing as a Symptom of General Paralysis', Lancet, Nov 13, 1875, pp 693 -5; on p 693. A taste of the disputes can be gleaned from numerous articles over the period; for example: J Burgess, 'The Policy and Pathology of Insanity', Lancet, 1851, II, pp 176 - 7; 246 - 7; 389 - 90; and 1852, I, pp 191-3; 305 -6; 539 -40; F Winslow, Lettsomian Lectures: On Insanity (London: John Churchill, 1854), Ch 3; Leader, Lancet, July 19, 1862, pp 66-7; Leader, Lancet, May 22, 1869, pp 718 -9; Leader, Lancet, July 19, 1873, pp 88 -9.

[51] Smith, 1981; Idem, 'Legal Frameworks for Psychiatry', in G E Berrios and H Freeman (eds), 150 Years of British Psychiatry (London: The Royal College of Psychiatrists, 1991), pp 137-151.

concentrating upon proof of insanity in criminals, missed the point of the legal M'Naghten rules: that proof of insanity did not in itself constitute lack of responsibility. Nevertheless, it was proof of insanity itself which alienists continued to address, primarily by positing a physicalist interpretation of human behaviour; and by arguing that they were peculiarly and solely qualified to judge the presence of mental disease.

The crime most associated with the general paralytic was larceny, since his classically grandiose delusions often made him believe that he owned everything around him. He had a reputation, too, for being a foolish and unmalicious thief who would make no attempt to conceal his crimes, and who would hand back stolen items with good humour if challenged. In keeping with alienists' preoccupations, their arguments focused upon the early stages of GPI; specifically the assumption that a patient with subtle physical signs of the disease must be already insane, and therefore irresponsible for his actions. Alienists clearly used a variety of strategies to persuade courts that patients they examined were mentally unsound; but, in contrast to categories such as impulsive or moral insanity, GPI offered a most convincing physical proof.[52] Tuke's legal position followed clearly from his stance upon the identity of GPI. Should a person with physical symptoms of the disease, he asked, be able to use the insanity defence despite an apparent absence of symptoms of mental disorder? The answer was clearly 'yes': 'It will be at once seen of what momentous importance it may become that the acts of a patient labouring under the physical symptoms of general paralysis, should not be judged by the same standard as is

applied to healthy brains ... I think the presumption should be in all these cases, that mental alienation had long existed, the former history of the patient should be studied, eccentricities of conduct, even acts of crime should be impartially examined, and if irreconcilable with the previous bearing and character of the patient, they should not be too harshly condemned as the acts of a responsible agent.'[53]

Numerous alienists reiterated this point. Clouston, commenting on the legal question in 1875, urged: 'We must not break the unity of a true pathological species through any merely accidental or legal definitions that set upon insanity as essentially different from other diseases of the nervous system.'[54] He described the case of a man with subtle physical signs of GPI: 'Anyone seeing him and speaking with him for a short time would be able to discover no mental defect whatever ... Yet are his mental operations not impaired? They are so, most evidently ... This man is clearly not 'insane' in any sense of the term, legal or even medical; yet are not his brain cells that minister to his mental operations being interfered with in their action by a disease that is only too sure still further to invade, and at last to abrogate, their functions? Should we be justified by any pathological rule in separating this case in any way from the other cases of the same disease where there are delusions, and who are reckoned insane?'[55] Alienists, it should be stressed, were not here talking about a spread of disease from physical to mental centres; for this stance would have clearly allowed a patient with physical signs of GPI to be as yet sane and therefore responsible. They were assuming that the mental affection already existed because the

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[52] See Smith's discussion of attempted proofs of insanity; Smith, 1981, pp 60 - 63.
[53] H Tuke, 1859, p 427.
[54] Clouston, 1875, p 191.

physical did; a philosophical position which was central to their plea for a naturalistic approach to criminal behaviour.

Maudsley's use of GPI in the legal debate stressed more explicitly the special qualification of the alienist - rather than the lawyer or the doctor with 'common sense' knowledge - to make the diagnosis of insanity. Maudsley, perhaps the most prominent alienist of the Victorian era, became Professor of Medical Jurisprudence at University College Hospital during 1869, and argued vociferously for his profession's authority. He gave histories of hitherto exemplary men who had given way to vice and appeared in court charged with assault or stealing; he stressed how the competent alienist detected a slight peculiarity of speech or inequality of the pupils, and was thus able to give a correct diagnosis of his insanity: 'Our knowledge is so exact that we can do what is the best test of a science - predict with certainty what will happen ... Plainly, common sense without special experience could have small chance of coming to a right conclusion in such a case.'[56] James Crichton-Browne's experience in the court-rooms typified for Maudsley the derision with which lawyers regarded the opinion of doctors: 'I shall not easily forget', Browne related, 'the smiles of incredulity which pervaded the Doncaster court-house, from the Recorder downwards, when I described the inequality of the pupils, slight tremor of the tongue, and other little symptoms which enabled me to recognise ... general paralysis'[57]

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[55] Ibid, p 191.

[56] H Maudsley, Responsibility in Mental Disease, (London: Henry S King & Co, 1874), pp 74 - 75; see also p 78: 'The theft in the early stages of general paralysis is a sufficienctly palpable fact; who but a physician familiar with the disease can recognise the inequality of pupils and the peculiarity of articulation which mark the beginning of incurable brain-disease, and give the true interpretation of the theft?'

[57] Quoted in ibid, p 694.

Maudsley continued to insist that the identity of GPI as a natural disease entity with a predictable course gave great weight to the credibility which should be afforded the expert alienist. But the problem of the insanity plea was also seen to reflect the poor education of all doctors in mental medicine - a further indicator of the poor status and isolation of the specialty. Prison doctors, who usually had the opportunity to observe patients over longer periods than did medical examiners, were rarely specially trained in mental disease. The alienist Wilkie Burman, for example, reported six cases in which general paralytics had been wrongly imprisoned for stealing; his criticism was directed at the prison medical officers who failed to recognise the signs of madness in their new charges. All doctors, he stressed, should have appropriate training; criminal care doctors in particular needed a special knowledge of insanity.[58] The necessity for the early recognition of GPI both within and outside court-rooms lay at the heart of numerous pleas for the medical corporations to institute formal teaching in mental diseases for all doctors. Frustratingly, though, the main role of GPI in these debates remained rhetorical. Although the disease was used by alienists to argue for their own conception of responsibility, there were not huge numbers of general paralytics charged with crimes, and those who were had usually committed minor offences rather than atrocities which captured the public attention. Despite the efforts of doctors, there appeared to be little change in the condescension which they received from the legal profession; and little change in their input into legal decision-making.[59] Advances in the teaching

[58] J Wilkie Burman, 'On Larceny, as Committed by Patients in the Earlier Stages of General Paralysis', J Ment Sci, 1873, 18, pp 536 - 543.
[59] See Smith, 1981.

of psychiatry, too, would be a long time in coming: undergraduate teaching would not become compulsory until the 1890's, and the Medico-Psychological Association's postgraduate Certificate of Proficiency in Psychological Medicine, instituted in 1886, would meet with only moderate success.[60]

GPI and the Science of Mental Disease

As alienists urged that GPI exemplified the unity of physical and mental, so they increasingly held it up as the best scientific model of mental disease. Through the 1860's and 1870's, members of the profession continued a long preoccupation with the possibility of a 'natural', scientifically-based psychiatric classification rather than one based upon 'mere symptomatology' - as had been proposed by Esquirol. Pathology, it seemed, would not provide this basis. In 1858, Tuke conceded: ' ... We are not yet in a position as regards our knowledge of the morbid appearances of the brain, to base our nosology upon the revelations of the dead-house. We can only wait an advance of knowledge which will render such a classification possible.' Such an advance did not materialise, and in the case of GPI the search for a characteristic brain lesion remained futile: in the 1870's, Blandford could state: 'Every portion of the brain has been thought to be the part affected ... The history of the investigation of general paralysis is this: observer after observer has found some morbid appearance which he has thought pathognomic of the disease, but which has been found to exist in the brains of other insane patients, or even in the brains of those not insane.'[61]

[60] See Bynum in Berrios and Freeman, 1991, pp 176-7.
[61] Blandford 1871, pp 286-7.

Although the dogma remained that GPI might prove the organic insanity par excellence, alienists turned again to the natural history of the disease - the collection of features which gave it a unique identity. GPI was consistently spoken of as the best model for this classificatory ideal, since it cut across many mental symptoms, unfolding as a clear and relatively predictable disease entity.[62] David Skae, alienist at the Edinburgh Morningside Asylum, made proposals which formed the basis for many classificatory schemes during this period - although their obvious flaws and inconsistencies were constantly debated.[63] His aetiological scheme, championed by his rather personally-biased former pupil Thomas Clouston, was founded on rigorous physicalism - in which 'everything mental or psychical connected with insanity' was excluded.[64] Skae presented GPI as the model for his 'rational and practical method of classification', since '... its natural history, including its symptomatology, progress, terminations, and pathology, are perhaps more complete than that of any other form of insanity.'[65] Maudsley, who applauded Skae's motives, was a prime spokesman for such a naturalistic conception of mental disease and rational classification. General paralysis was, he claimed, the one exception to the 'purely psychological ... vague, artificial, and unsatisfactory' classification of mental disease - the prime example of

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[62] The French historian Bercherie regarded Bayle's depiction of a disease cutting across the symptomatic nosologies of Pinel and Esquirol as extremely radical (far more so than its anatomical basis); see Bercherie, 1980, esp pp 75-6. Berrios also stresses this point, as a refutation of GPI's importance in terms of anatamo-pathology: Berrios, 1985, pp 396-7; 1995, p 39.

[63] D Skae, 'A Rational and Practical Classification of Insanity', J Ment Sci, 1863, 9, pp 309 - 319. The classification was presented to the Association of Medical Officers of Asylums and Hospitals for the Insane during this year. Maudsley, Bucknill, Hack Tuke and Blandford all applauded Skae's classification or incorporated it into their own classifications.

[64] J C Bucknill, 'A New Classification of Insanity', Lancet, Nov 15, 1873, pp 696 - 697; on p 696. See A Beveridge, 'Thomas Clouston and the Edinburgh School of Psychiatry', in Berrios and Freeman, 1991, pp 359 - 388; esp p 370-1.

future possibilities, and of 'the revolution which seems impending'[66] He continued to cite GPI in his pleas for a scientific classification for the next fifteen years. The disease gave, he maintained, 'the strongest practical condemnation to a purely psychological classification ... We have only to do with other forms of insanity as we have done with general paralysis ... to arrive at a natural classification of them.'[67]

Clouston similarly claimed GPI as the key to the future. During 1875 he defended Skae's classification against the numerous criticisms of Crichton-Browne, who claimed that Skae's Morningside school, opposing the study of mental symptoms per se, had a stranglehold on scientific opinion. Crichton-Browne, who placed great emphasis upon the psychological aspects of mental science, derided dreams of 'mounting a delusion in Canada balsam or ... detecting despondency in a test tube', and held that alienists must be content at present simply to understand symptomatology.[68] Clouston, in typically fulsome language, used GPI as the test case against Browne: 'Does he deny that General Paralysis ... is a true cerebro-mental disease, a distinct clinical, symptomatological, and pathological reality? ... The most distinct, the most real, the most undisputed, the truest cerebromental disease ... cannot be provided for in [Esquirol's symptomatic] classification that he [Browne] defends ... Can anything more powerful be urged against our accepting it as final; or any stronger incentive be applied for us to invent a better?'[69] Clouston had great faith that other

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[65] Skae, 1863, p 314. Epilepsy and puerperal mania were regarded as less perfect models of natural disease.
[66] H Maudsley, 'Illustrations of a Variety of Insanity', J Ment Sci, 1868, 14, pp 149 - 162; 149; 151.
[67] H Maudsley, 1874, p 76.
[68] Quoted in T S Clouston, 'Skae's Classification of Mental Disease', J Ment Sci, 1875 -6, 12, pp 532 - 550; on p 533.
[69] Ibid, p 536.

mental diseases would be revealed to be as unique as GPI: 'Did we know everything about general paralysis and epilepsy, we should find the path of research into most other diseases of the nervous system comparatively easy. They would be the key to all the rest ... It is quite certain that under the term insanity there are included many pathological species of brain disease, just as distinct as general paralysis, which we shall ultimately be able to segregate and distinguish.'[70]

Predictably again, GPI yielded no fruit in terms of improved classifications of insanity. Crichton-Browne's comments appeared well justified, as the majority of mental disorders simply appeared refractory to natural historical classifications. And ironically, those who were consciously trying to create a scientific psychiatry based on neurophysiological principles were those most antagonistic towards schemes such as Skae's, which ignored the psychological dimensions of mental classification. Thomas Laycock, Professor of the Practice of Medicine at Edinburgh, made a scathing attack of Skae and his school. He held up Crichton Browne as the prime exponent of 'high and true scientific culture', and urged that Skae's successor Clouston should carry on these principles in collaboration with himself at Edinburgh.[71] At the end of the century, the classification adopted by the Medico-Psychological Association (MPA) for use by asylum superintendents fell back upon traditional categories - using symptomatic descriptions for all disorders apart from GPI itself and epilepsy.[72]

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[70] Clouston, 1875, p 189.
[71] T Laycock, 'The Teaching and Practice of Psychological Medicine as Influenced by Classifications of Insanity', Lancet, Jan 3, 1874, pp 4 - 6; Jan 10, 1874, pp 48 - 50; on p 50.
[72] W F Bynum, 'Tuke's Dictionary and Psychiatry at the Turn of the Century', in Berrios and Freeman, 1991, pp 163 - 179.

Notwithstanding this failure, GPI seemed to hold great potential for opening the doors of neurophysiology and neurology to alienists. Clinical histories of GPI during the middle decades of the century included hosts of symptoms and signs which, increasingly, gave the disorder a neurological flavour: abnormalities of limb sensation; incoordination; loss of reflexes; pupillary changes; cranial nerve abnormalities causing peculiar facial features such as teeth-grinding; and epileptiform fits. Interest in such physical features, and the use of neurological techniques to investigate them, was commonplace in psychiatric research from the 1840's - although such techniques would not be applied to asylum practice until the 1890's. Shortly after GPI was first described, attention was focused on the nature of the paralysis involved. Electro-galvanic experiments during the 1850's established that motor excitability was normal in the limbs of patients, and this finding was corroborated by the observation that apparently paralysed patients could move their limbs in moments of stress. By the end of the decade, therefore, it was widely accepted that this was not a 'true' peripheral paralysis, but rather a central paralysis - a loss of 'directive power' in the brain.[73] Here was a convincingly scientific corroboration of the philosophical position which alienists were so eager to present: the unity of the physical and the mental.

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[73] Bucknill published electro-galvanic experiments in 1852 which, he claimed, demonstrated loss of motor excitability in the limbs of general paralytics: his stance supported his view that GPI was a nutritional disease of the whole nervous system, and not just the brain: reported in Bucknill and Tuke, 1858, p 328. However, this position was soon opposed by consensus: see, for example, Skae, 1859-60; and E Salomon, 'On the Pathological Elements of General Paresis or Paresifying Mental Disease (Paralysie générale)', transl from the Swedish by W D Moore, J Ment Sci, 1862, 8, pp 365 - 385; on p 376: 'Paralysis proceeds from the brain ... patients can, when delirious, use the muscles very violently ...'

However, the appearance of a newly-defined disease 'tabes dorsalis', threatened to complicate this brain-centred picture of GPI. Tabes dorsalis - a disease entity crystallised from a diffuse and long-recognised group of spinal symptoms classed 'tabes' - entered the clinical scene during the 1840's, primarily as a result of work by the emerging German neurological school. Horn and Romberg's intensive clinico-pathological approach provided a rapid consensus of opinion as to the characteristics of the disease: and in contrast to GPI, a specific pathological lesion (degeneration and inflammation of the dorsal columns of the lower spinal cord) was associated with specific clinical features with little debate.[74] The classic symptoms included incoordination of the legs, causing a characteristic swaying and tottering gait (accentuated by asking the patient to close his eyes); excruciating 'lightning' pains throughout the body; sexual and urinary dysfunction; and multiple forms of joint disease elucidated by Charcot during the 1870's.[75] In 1871 the German psychiatrist Westphal also suggested that the loss of the patellar tendon reflex was practically diagnostic of the disease. Tabes ran a protracted course, and often resulted in complete loss of movement, gangrene, and death. The two interchangeable names for the condition reflected the equal importance afforded to the clinical and pathological features: 'tabes dorsalis' referred to the characteristic pathological lesion (wasting of the spine), whilst the French neurologist Duchenne's term 'locomotor ataxia' referred to the corresponding clinical syndrome.

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[74] Schiller, 1976.

[75] See Romberg's classic account: M H Romberg, A Manual of Nervous Diseases of Man, transl E H Sieveking , 2 Vols (London: Sydenham Society, 1853), Vol 2, pp 395 - 401.

Insanity did not enter into the definition of tabes dorsalis; nevertheless, the newly delineated disease bore an interesting relationship to GPI. Both involved similar movement disorders, often affected the cranial nerves, and had a progressive and fatal nature. These apparent links between the two diseases were strengthened by the observation of a common spinal pathology. In 1857, Rokitansky described characteristic tabic spinal pathology in cases of GPI, and this observation became increasingly frequent until Westphal could state in 1864 that such spinal features were common, if not constantly present.[76] Westphal also suggested a clinical link between the two diseases. He observed that there was a form of tabes dorsalis which, many years after its onset, bore features of insanity characteristic of GPI.[77]

It would be stretching the point to say that this puzzling common ground was a major preoccupation or bargaining point for alienists. A traditional retrospective interpretation would suggest that cases which alienists claimed were GPI without insanity were in fact cases of tabes; and that cases claimed to be tabes terminating in insanity were in fact GPI.[78] In most cases, however, the overall physical features of each disease were considered clear enough to differentiate them - without needing to use the presence or absence of insanity as the defining factor.[79] There is no obvious evidence that alienists tried to 'claim' cases which neurologists would have defined as

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[76] C Westphal, 'Cases of tabes dorsalis (grey degeneration of the posterior columns) and paralysis universalis progressiva', trans J F Rutherford, J Ment Sci, 1864, 10, pp 207 - 220; see also Bucknill and Tuke, 1858, p 328.

[77] Westphal, 1864; Idem, 1868.

[78] See, for example, Zilboorg and Henry, 1941.

[79] See, for example, Gairdner, who described GPI without insanity, but noted that there was 'no difficulty in differentiating from locomotor ataxia', since there were no lightning pains, nor a stamping gait: Gairdner, 1876, p 255. Similarly doctors discussing tabes terminating with mental symptoms might conclude that the disease was 'simulating' GPI, but was clearly not GPI: J W Plaxton, 'An

tabes, by arguing well-hidden or incipient insanity. It can rather be argued that alienists were initially quite resistant to acknowledging the spinal aspects of GPI, since this represented a threat to the picture of a disease in which the physical and the mental were unified in the brain pathology. Until the 1870's it was rare for alienists to take much interest in the spinal pathology of GPI: although Bucknill mentioned in passing the frequent finding of spinal atrophy in 1858, he did not cite it in connection with tabes, but as evidence for his theory that GPI was due to diseased nutrition of the whole nervous system.[80] Ten years later the alienist Boyd mentioned that he had frequently seen spinal cord changes in GPI but that, curiously, no other English author had written about them.[81] He attributed this to the difficulty of exposing the spinal cord during post-mortem; but others did not discount the possibility of ideological motives. Westphal charged his psychiatric colleagues with persistently overlooking GPI's possible connection with spinal disease, since they preferred to view it purely as a brain disease: 'Thus it came to pass that in the framing of theories regarding the nature of the paralysis, the spinal cord was either entirely ignored, or the purely cerebral character of the disease was ... emphatically inculcated as distinguishing it from other spinal affections ...'[82] To Westphal, tabes and GPI could no longer be regarded as independent diseases; and he used this pathological point as a metaphor for the relationship between the specialties of neurology and psychiatry: '[I aim to] ... unite the phenomena observed in the asylum with ascertained facts in nervous

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Account of Two Cases of Locomotor Ataxia, with Mental Symptoms Simulating those of General Paralysis', J Ment Sci, 1878, 24, pp 274 - 278.

[80] Bucknill and Tuke, 1858.

[81] R Boyd, 'Observations on General Paralysis of the Insane and on the Morbid Changes found on Post-Mortem Examination in the Spinal Cord', J Ment Sci, 1871, 17, pp 1 - 24; 364 - 370.

pathology, and to reconcile them with each other ... The time is, I believe, at hand when ... the veil of the asylum will be removed, when once for all the barriers will be thrown down which divide mental pathology, as we must still call it, from that of the remainder of the nervous system.'[83]

The alienist Blandford made a similar charge of his British colleagues in 1871: most of his specialty, he claimed, only described GPI changes in the brain, 'which they consider to be the seat of the disease' - whilst neurologists focused upon the spinal cord changes of their own disease tabes.[84] In 1878 Wilks - a specialist in diseases of the nervous system at Guy's Hospital - noted the wasting of both cerebral and spinal centres in GPI, and echoed Westphal in linking this to the relationship between the two specialties: 'It may appear to you ... that I am going now out of the wards of the hospital to the madhouse; but, in fact, I am following the natural order of disease, for the forcible separation of many cases found in lunatic asylums from those which we have to treat here is artificial ...'; 'There are ... cases of disease commencing in the spine, and subsequently creeping up to the head, and which, strictly speaking, are cases of general paralysis ... I believe it is now generally admitted that patients who have had symptoms in the first place only of locomotor ataxia have ended with GPI ... An alienist physician could not allow, however, that the condition was the same, for it would contradict his strict rule that the brain, or at least the mind, is first affected.'[85] He posed the opposite legal position from that of the majority of alienists,

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[82] Westphal 1868, p 506, see also p 512.
[83] Ibid, p 163.
[84] Blandford, 1871, p 286.
[85] S Wilks, Lectures on Diseases of the Nervous System (London: J&A Churchill, 1878), p 163; p 171.

and advised withholding the term 'GPI' even if the disease strictly was - since the term suggested a patient was mentally incapacitated from the first.

Wilk's latter point suggested why alienists may have been reluctant to countenance the spread of a spinal to a cerebral disease: such a position undermined the assumption that the physical and mental symptoms were united by cerebral pathology - and thus that a patient with GPI was necessarily already insane. From the late 1870's, however, doctors would turn their attention to the possible syphilitic aetiology common to both diseases - and this new criterion of definition would provide a compelling reason to unite them clinically. Gowers spoke for alienists as well as for his neurological colleagues when he claimed in 1886 that GPI was as much spinal as cerebral, that the two diseases sometimes changed from one to the other, and that they differed by preponderance of symptoms rather than by absolute distinction: 'It is probable that syphilis predisposes to general paralysis as well as to tabes. They are often combined ... It may be difficult to say in which category a case should be placed.'[86] Some figures claimed that as many as 10% of tabetics became general paralytics, and that at least a third of general paralytics presented with tabetic signs. With the common aetiology established towards the end of the century, Alfred Fournier - champion of the syphilitic cause of both diseases - would feel confident in ascribing them 'two topographic modalities of a single and same morbid process', both of which could be subsumed under his new category of 'parasyphilis'.[87]

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[86] W R Gowers, Lectures on the Diagnosis of Diseases of the Brain, 2e (London: Churchill, 1887), p 243.

[87] A Fournier, Les Affections Parasyphilitiques (Paris: Rueff et Cie, Éditeurs, 1894), p 231. See Chapter 2 below.

Nevertheless a pathological union would not unite the two diseases in practice, since pragmatic issues of patient care remained far more important than theory. Between 1860 and 1890, a group of doctors succeeded in establishing neurology as a specialty practice, based at the National Hospital for the Paralysed and Epileptic at Queen Square and the Hospital for Epilepsy and Paralysis at Maida Vale.[88] The contrast between facilities available to the new neurologists and to alienists was painfully clear, although friction was rarely overtly expressed. At the National Hospital, opened in 1860, all potential admissions were first seen as out-patients and selected on medical grounds: 'Difficult and dirty, unmanageable and incurable [patients] were excluded by the bye-laws.'[89] The hospital was staffed by motivated voluntary doctors who had full-time appointments elsewhere, and the doctor-patient ratio was roughly 1 to 6. The contrasting conditions in public asylums were obvious; and the practical separation of the two specialties has been noted by historians.[90] The case of GPI and tabes demonstrated perfectly this gulf, despite the promising neurological aspects of the alienists' disease. Neurologists took it for granted that they would care for tabetics - but that if inappropriate symptoms intervened, they would relinquish this care. Thus clinical notes for the National Hospital documented a

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[88] The creation of British neurology has received little attention from historians. For traditional accounts, see G Holmes, The National Hospital, Queen Square, 1860 - 1948 (Edinburgh and London: E&S Livingstone Ltd, 1954); M Critchley, 'The Beginnings of the National Hospital, Queen Square (1859 - 1860) in Idem, The Black Hole and Other Essays (London: Pitman Medical Publishing Company, 1964)

[89] R Hunter and I Macalpine, Psychiatry for the Poor (Folkestone, Wm: Dawson and Sons Ltd, 1974), p 203. The authors noted the disgruntled comment of a visiting asylum superintendent: 'At the National, doctors discarded the unrewarding and incurable, at Colney Hatch they lived with them ...', p 203.

[90] 'Neuropsychiatry' never flourished in Britain as in Germany, notes Bynum, although 'lip-service was paid to the notion of the organic nature of insanity ...': Bynum, 1985, p 90. Hunter and Macalpine, themselves physicalist psychiatrists, referred to this separation with some bitterness: Hunter and Macalpine, 1974, p 205.

handful of cases annually in which those with equivocal tabes were admitted, subsequently became insane or caused disturbance on the wards, and were promptly removed back to relatives or transferred to an asylum.[91] Correspondingly, GPI appeared only in passing in neurological textbooks.[92] Alienists in their turn accepted their lot of caring for the overtly insane - and had little practical leeway for claiming patients other than those confined to asylums during this period.

Bynum notes that the two specialties did cross-fertilise to a certain extent after the 1870's - but GPI demonstrated the necessary limits of this collaboration. Jacyna's suggestion that alienists did no more than toy with neurophysiological principles during the middle decades - using them to 'place a thin veneer of modernism' over old approaches - seems in many ways to hold true for the latter part of the century.[93] For many alienists writing about GPI - who used such imagery as central nervous 'force' or 'reserve' - the cerebral nature of the disease remained a corroboration of a useful philosophical position, rather than a fertile field for the use of neurophysiological principles. This could be said of Henry Maudsley who, partly influenced by Laycock, attempted to apply reflex neurophysiological principles to psychiatry. To him the interest of GPI lay in the central (rather than peripheral) nature of its paralysis, which demonstrated the close analogy between mental ideas and intelligent power over muscles: 'What is further diseased [in GPI] is the region of the motor intuitions or actuation - the nervous centres in which the motor residua are organised, rather than muscular power ... those motor residua are affected which are

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[91] Clinical case-books for the National Hospital, Queen Square (1870 - 1877; 1883 - 1885). Archives of the National Hospital, Queen Square.

[92] There was only brief mention, for example, in Gower's 1892-3 manual of nervous diseases.

in the closest relations with the intellectual life ...'[94] GPI demonstrated that the physical and mental were so closely allied 'that we could not think without some means of physical expression ...'; thus the gradual failure of power of movement might actually aggravate the mental decay.[95] Such suggestions remained philosophical, and did not contribute in any concrete way to neurophysiological advances.[96] And whilst Maudsley's pronouncements seemed to many of his profession dogmatic and overly philosophical, alienists continued to bemoan a lack of fruitful scientific research in their field - mainly because of the burdensome administrative duties of asylum doctors.[97]

At the West Riding asylum in Wakefield, James Crichton-Browne tried to counter the 'vindictive attacks which have been recently directed against the public lunatic asylums of this country' due to a putative lack of practical scientific interest.[98] Crichton-Browne, Edinburgh-born son of the alienist W A F Browne, greatly admired Laycock, and attempted to apply his work on cerebral physiology throughout his psychiatric career. Between 1871 and 1876 he hosted research programmes and annual scientific 'conversazioni', in collaboration with general physicians such as Clifford Allbutt and doctors specialising in neurology such as Hughlings Jackson.[99]

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[93] Jacyna, 1982.
[94] H Maudsley The Physiology and Pathology of the Mind, 2e (London: Macmillan & Co, 1868), p 364.
[95] Ibid, p 365.
[96] See Jacyna, 1982, who questions the practical importance of Maudsley's neurophysiological stance, and suggests that he continued to place far more emphasis upon the old principles of organicism.
[97] For Maudsley's reputation amongst his colleagues see M Collie, Henry Maudsley: Victorian Psychiatrist (St Paul's Bibliographies: Winchester, England, 1988).
[98] J Crichton Browne (ed), The West Riding Lunatic Asylum Medical Reports, 1871, 1, Preface.
[99] See M Neve and T Turner, 'What the Doctor Thought and Did: Sir James Crichton-Browne (1840 - 1938), Med Hist, 1995, 39, pp 399 - 432; H R Viets, 'West Riding 1871 - 1876', Johns Hopkins

The resulting copious medical reports were far more neurologically orientated that the usual research output from asylums - indeed than the typical content of the Journal of Mental Science. Research concentrated upon the use of instruments such as the ophthalmoscope and sphygmograph; detailed histological studies of the brain; experimental pharmacology; and experimental neurophysiology. In particular, David Ferrier used Browne's facilities to research and publish his early influential study into cerebral localisation - an experimental follow-up both to Hughlings Jackson's clinical work upon epilepsy, and to Fritsch and Hitzig's galvanic stimulation studies.[100]

GPI was one focus of attention at the West Riding, and a number of papers appeared which carried the authentic stamp of science, including ophthalmological, laryngoscopic, urinological, and cardiac observations, and histological studies of the cortex and cranial and peripheral nerves.[101] As the new neurologists increasingly turned their attention to cerebral localisation, GPI became alienists' talisman for cerebral research in this field, and particularly for the possibilities of symptom-pathology correlation: 'The most interesting field of pathological enquiry in this disease', wrote Clouston in 1875, 'is the tracing of a definite connection between the symptoms present during life and the amount of disease present in the different parts of the central nervous system. Considering how the symptoms, motor and mental, are often so limited and special, we have here a wonderful field of physiological as well

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Institute History of Medicine Bulletin, 1938, 6 (1), pp 477 - 487; J Todd and L Ashworth, 'The West Riding Asylum and James Crichton-Browne, 1818 - 76', in Berrios and Freeman, 1991, pp 389 - 418.

[100] D Ferrier, 'Experimental Researches in Cerebral Physiology and Pathology', in The West Riding Lunatic Asylum Medical Reports, 1873, 3, pp 30 - 96.

[101] See, for example, C Aldridge, 'Ophthalmoscopic Observations in General Paralysis', in West Riding ..., 1872, 2, pp 223 - 253; J Merson, 'The Urinology of General Paralysis', in West Riding ..., 1874, 4, pp 63 - 93; L Browne, 'Laryngoscopic Observations in General Paralysis', in West Riding ..., 1875, 15,

as pathological observation. No experiments in injuring or faradising limited portions of the brain could be more perfect than what occurs in this disease'[102] Such pleas to correlate symptoms with localised pathology along the lines of Ferrier were often made during the 1870's.[103] Yet as neurologists continued to make high-profile strides in research, these hopes never seemed to materialise for GPI. Epilepsy proved itself far more amenable to neurophysiological research, and as the focus of Hughlings Jackson's work and the commonest reason for admission to specialist hospitals such as the National, it quickly became the centrepiece of the new neurology.[104] Meanwhile mental features in GPI simply could not be related to specific brain areas; and studies such as Newcombe's on epileptiform seizures threw more light upon epilepsy itself than upon GPI.[105]

Crichton Browne's experience as the high days of West Riding came to an end was that the disease was as imperfectly understood as ever, and moreover was increasingly met with extreme fatalism by his colleagues.[106] West Riding would gain its later reputation as a birth-place for neurology rather than as a stimulant for psychiatry. As Crichton-Browne moved on to co-found the journal Brain with Bucknill, Ferrier, and Hughlings-Jackson, and as Maudsley was ousted from the

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pp 271 - 283; J M Fothergill, 'The Heart Sounds in General Paralysis of the Insane', in West Riding ..., 1873, 4, pp 94 - 151.

[102] Clouston, 1875, p 201.

[103] See, for example, I Ashe, 'Some Observations on General Paralysis', J Ment Sci, 1876, 22, pp 82 - 91; and Mickle, 1886, p 407. Mickle believed that GPI would elucidate both the linking of symptoms to areas of critical pathology, and the physiological differences between the cerebral hemispheres.

[104] J Hughlings Jackson, Selected Writings, ed J Taylor (London: Staples Press, 1958), 2 Vols. Ironically Jackson carried out many of his studies upon epilepsy which was secondary to cerebral syphilis - a syndrome which would bear a controversial relationship to GPI: See Chapter 2 below.

[105] C F Newcombe, 'Epileptiform Seizures in General Paralysis', in West Riding ..., 1875, 5, pp 198 - 226.

Journal of Mental Science to make way for a less discursive, more rigorously clinical approach, the scene was again set to reassert science in psychiatry. Brain, founded in 1876, aimed to unite neurology and psychiatry by covering the physiology and pathology of the nervous system as a whole: 'mental phenomena will be treated only in correlation with their anatomical substrata, and mental disease will be investigated as far as possible by the methods applicable to nervous diseases in general.'[107] Hopes for a fruitful collaboration, however, did not seem to be borne out by the content of the journal. Contributions remained overwhelmingly biased in favour of neurologists: tabes dorsalis, for example, appeared in many articles over the first ten years, whilst GPI appeared in only a handful.[108] Those articles by alienists were often reprints of articles appearing elsewhere, or book reviews; and rather than original research in psychiatry the journal was often a forum for the usual complaints about the poor state of the discipline, and reassertions that insanity was a physical disease.[109] By 1880 Crichton-Browne was hardly optimistic about the quality of research in public asylums: '... Our scheme of mind doesn't harmonise with any scheme of brain, and our exploration is not in accordance with knowledge of cerebral topography ...' It was essential, he again urged, to link symptoms with recent research into cerebral localisation.[110] Three years later, a comment of Clouston summarised the paradoxical mixture of faith and disappointment which GPI elicited: 'Its study has somatised and

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[106] J Crichton Browne, 'Notes on the Pathology of General Paralysis of the Insane', in West Riding ..., 1876, Vol 6, pp 170 - 231.
[107] Brain: A Journal of Neurology, 1878, 1, Introduction.
[108] Of about 25 contributions in the first publication, only 6 were by alienists.
[109] See, for example, J C Bucknill, 'The Late Lord Chief Justice of England on Lunacy', Brain, 1881, 4, pp 1 - 26.
[110] J Crichton-Browne, 'A Plea for the Minute Study of Mania', Brain, 1880, 3, pp 347 - 362; on p 340.

definitised [sic] the study of all mental diseases, and has added, and will add still more, to our knowledge of the connection of mind with body, and of mental and motor disturbances ... [But] what we knew of its symptoms and pathology ought to have led to the conclusion that the cerebral convolutions have motor functions long before Hughlings Jackson, Hitzig, and Ferrier arrived at their generalisations on the subject.'[111]

Alienists then - as so often - felt themselves on the sidelines of progress, lamenting lost chances. Whilst describing GPI as a key to the potential of mental science, they remained painfully aware that their rhetoric was not being translated into theoretical developments or practical benefits - just as 'the promise of the mid-century foundations remained unfulfilled' for the specialty as a whole.[112] Meanwhile, the numbers of the disease's victims appeared to rise inexorably, and its demeaning course emphasised the sorry picture which overcrowded asylums presented. By the 1880's a mid-century view of the disease still seemed apt: 'GPI must be regarded as a rich unexplored mine of physico-psychical curiosities, [rather] than as a curable malady...'[113] Hope and interest, however, was being rekindled as alienists turned their attention to a new facet of the disease: its cause. The possibility of a link with syphilis was providing a fresh and potentially far more fruitful avenue for alienists to explore; and as the century drew to a close it was also contributing rich new strands of imagery to an appalling form of insanity.

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[111] T S Clouston: Clinical Lectures on Mental Diseases (London: J&A Churchill, 1883), p 354.
[112] Bynum in Berrios and Freeman, 1991, p 177.
[113] 'Review of Austin 1859', Lancet, Nov 5, 1859, p 463.

CHAPTER 2

MASCULINITY, IMMORALITY, and SYPHILIS: THE CAUSES OF GPI

Introduction

In 1892 the Edinburgh asylum physician Wilson summarised the typical character of the general paralytic:

'The life record of some of these men reads like a novel by Charles Lever - brimful of incident, stress, and struggle ... The characteristic general paralytic is a man with a large belief in himself, restless, ambitious, and with a relentless desire for the good things of this life ... the making and marring of more than one or two of his lady friends may be taken for granted ... [He is] impulsively generous, good-natured and generally companionable ... Intelligence and common sense, ambition, energy, sociability and a large capacity for enjoyment, a firm belief in oneself and a preference for handsome women are all eminently sane characteristics according to our present standard.'[1]

Compare a contemporary portrayal by the Glamorgan asylum physician Stewart, commenting upon an apparent rise in GPI:

'This ominous feature of the life of the nation is not related to the altruistic, other-regarding instinct ... Selfish indulgence, lustful gratification, insatiable animalism, "general sensuality and fastness," - these are to a very large extent the

[1] G R Wilson, 'The Diathesis of General Paralysis', J Ment Sci, 1892, 38, pp 30 - 41; on pp 37, 40.

"grand parent-manufactory of the evil" ... General paralysis ... is the apotheosis of selfishness. The opening chapter is moral decadence; the closing acutely rapid physical and intellectual degeneration and inevitable premature extinction.'[2]

These two accounts, one colourful and affectionate, the other full of horror, demonstrate an enduring ambivalence in portraying the moral character of the general paralytic during the nineteenth century. Whilst both described the egotistical man prone to excesses such as overwork and sexual activity, each drew out contrasting aspects of this egotism which cast the potential patient as either admirable or disgusting. The theme of excess was, of course, a common preoccupation in Victorian psychiatry. The conflict, for example, between creditable work and potentially destructive overwork was never easily resolved; and asylum records from the early nineteenth century onwards reflected this as they recounted numerous cases of industrious, sober men succumbing to a variety of insanities because of anxiety and business reverses. The work-overwork conflict intensified during the latter half of the century when unease about the frenetic demands of civilisation crystallised into fears of the threat of degeneration. In particular neurasthenia - seen at best as an indicator of society's malaise and at worst as the first step towards degenerative perdition - was explicitly linked to intellectual over-ambition amongst the middle and professional classes.[3] Alcoholic and sexual excess too were routinely mentioned in connection with all forms of insanity during the Victorian period; and were increasingly used as

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[2] R S Stewart, 'The Increase of General Paralysis in England and Wales: Its Causation and Significance', J Ment Sci, 1896, 42, pp 760 - 777; on p 776.

emblems of mental disintegration as the century progressed. Once again these particular evils featured regularly in discussions of degeneration, which frequently conflated inherited and socially acquired causes of racial decay.

GPI, however, demonstrated particularly clearly how ambivalent images could be attached to the themes of character and insanity. During the middle decades of the century alienists presented a very distinctive picture of the potential patient, emphasising his admirable masculine characteristics of vigour, energy, ambition, intelligence and endurance. This positive perception was surprisingly dominant despite an acknowledgement that physical incontinence also played an important part in the aetiology of the disease. It was, I would suggest, largely responsible for the fact that doctors never linked GPI particularly strongly with neuropathic heredity before the 1890's. Whilst both the positive and negative facets of excess would be continually reworked in medical writings, the balance between them clearly changed as the century progressed. This change occurred as GPI was increasingly linked with syphilis at the end of the nineteenth century. The making of the link has not been considered in any depth by historians - although it is mentioned in passing in a number of general accounts. The traditional secondary sources usually depict a sharp opposition between enlightened scientific progressivists who supported a syphilitic aetiology, and traditionalists who opposed the hypothesis - an opposition which many contemporary doctors themselves sought to present.[4] Such a polarisation is, of course, far too simplistic for this complex and fascinating episode in medical history;

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[3] See B Sicherman, 'The Uses of a Diagnosis: Doctors, Patients, and Neurasthenia', J Hist Med, 1977, 32, pp 33 - 54.

and a closer examination of the debates reveals the interplay between a variety of factors which might have influenced contemporary views.

At the most obvious level, doctors were grappling with medical evidence for the link: whether epidemiological, pathological, clinical, or statistical. Detailed medical discussions filled the journals for decades, and they reveal that what was at stake was the comparative validity of these different forms of evidence. The domain of syphilis had been traditionally defined by its pathological identity and by its response to standard therapy. GPI, however, did not fall within these criteria; instead, the link was to be argued for largely upon the basis of statistical evidence. Coupling the two diseases thus required a broadening perception of how syphilis was defined - a clinical shift which many British doctors accepted only cautiously. Changing perceptions about the clinical domain of syphilis were reflected by intense professional and public interest in the disease from the end of the nineteenth century - exemplified by the presentation of congenital syphilis as the archetypal degenerative threat. Closely bound to arguments about the definition of disease, then, we can identify social and professional concerns which might have influenced support for, or antagonism towards, linking GPI with syphilis. First, there are instances in which the relationship between emerging medical specialties was directly applied to clinical questions. This appears most clearly in the work of Alfred Fournier, the flamboyant French syphilographer who explicitly linked the widening domain of his chosen disease with the establishment and growth of venereology as a specialty. Such rhetoric was not as obvious in Britain, which lacked a similarly vociferous champion for

[4] See, for example Ackerknecht, 1959; Crissey and Parish, 1981; Quetel, 1990; Zilboorg and Henry,

venereology. Instead a self-consciously moderate response emerged, in which the weighing of clinical evidence was claimed to be paramount.

Second, I have argued that GPI was presented as a scientific model by nineteenth-century British alienists - however fruitless this proved to be in practice. We might suggest, therefore, that the syphilitic basis of GPI would be seized upon as a vindication of this ideal. This point has been considered by a number of historians - usually in the context of the early twentieth century. Lunbeck, commenting upon the 1910's in the United States, suggests that the formulation of 'neurosyphilis' had a huge impact for psychiatrists, since it distanced them from earlier moralistic theories of aetiology and underwrote their claim to scientific status: 'Syphilology brought psychiatry into medicine by means of the disease paradigm to which it conformed.'[5] Thomas Szasz has made a similar assumption in service of his own political stance towards psychiatry.[6] It is surprisingly hard, however, to detect such enthusiasm for syphilis as a status-symbol during the late nineteenth century - a point which Berrios has noted: 'If it is the case that nineteenth-century alienists were totally organic in their approach, why did they not accept the syphilitic aetiology as a gift from the gods?'[7] Berrios uses this apparent anomaly to support his thesis that GPI was not considered as a special or paradigmatic disease. I would suggest that - in Britain at least - caution was largely due to questions about the clinical identity of syphilis which I have introduced above. Although the link was generally accepted between

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[5] E Lunbeck, The Psychiatric Persuasion (Princeton, New Jersey: Princeton University Press, 1994), p 117.

[6] Szasz, 1976.

[7] Berrios, "'Depressive Pseudodementia' ...', 1985, p 398.

1880 and 1907, it could not be called a dramatic revolution - despite Fournier's rhetoric. Alienists continued to use multifactorial models of aetiology well into the twentieth century, so that syphilis was incorporated into traditional perceptions of GPI, rather than replacing them wholesale. More dramatic changes in aetiological perception would wait until the application of laboratory medicine during the first decades of the twentieth century.

Third, historians have quite naturally turned to the shared stock of ideas about morality, sexuality and degeneration which both syphilis and insanity suggested to Victorian observers. Porter and Berrios speak of 'the late nineteenth-century obsession with GPI' which was conditioned to a large extent by growing fears of the consequences of unbridled sexuality: 'Sooner or later ... all who had committed youthful sexual indiscretions would pay the price by growing demented.'[8] Showalter similarly highlights the conflation of moralistic interpretations of the two diseases at the end of the 1890's: 'The new understanding of general paralysis [as terminal syphilis] ... was the perfect confirmation of late Victorian psychiatrists' belief in heredity and visible vice ...'[9] In her study of Clouston, Thompson suggests that ideas about GPI - 'an irrevocable consequence of dissipated habits' - came from the same stock of ideas as those about syphilis and led to a heightened perception of such patients as 'increasingly diseased trouble makers and deviants.'[10]

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[8] Berrios and Porter, 1995, p 59.
[9] Showalter, 1986, p 89. See also Spongberg, 1998, p 238: 'The discovery that GPI was related to syphilis conclusively connected it with the accepted nosography of degeneration, hereditary tendency to vice and disease'.
[10] Thompson, 1985.

GPI demonstrates well how the concept of degeneration could be applied in nebulous ways. The majority of alienists were adamant that it was linked to heredity (whether neuropathic or syphilitic) far less commonly that were other forms of insanity; and that it appeared in manly but fast-living non-degenerates. On the other hand, it seems clear that Fournier's far-reaching concept of hereditary syphilis primed alienists to accept a link between venereal disease and insanity. From the 1880's onwards images of the disease became more obviously ambivalent as GPI took on the derogatory implications of the venereal taint. First, GPI came to epitomise a form of degeneration tragically acquired by the self-ruining man. Then, over the turn of the century many writers vividly represented GPI as the epitome of the collective evils of civilisation. Nevertheless, despite the assumptions of historians, the disease often retained surprisingly stubbornly its admirable overtones; and it remained overwhelmingly associated with hitherto valuable members of society, rather than the degenerate underclasses. Whilst female general paralytics were often portrayed as shadowy, colourless figures showing atypical forms of the disease, GPI remained essentially a firmly 'masculine' and 'middle-class' disease.

'Disease of Manhood'

Bayle, in his original account of GPI, noted two groups of sufferer: military men and the poorer classes who were subjected to physical privation with venereal and alcoholic excess; and ambitious middle-class men who undertook excessive mental and physical exertion in pursuit of their intellectual or business goals. Bayle strongly associated this latter class with the symptomatic category of ambitious monomania; a

point which was central to his disagreement with Esquirol concerning the status of 'arachnite chronique'. In fact, despite Esquirol's reluctance to annexe monomania to Bayle's new disease, he persisted himself in linking GPI closely with single-minded ambition; and described large numbers of cases in which young monomaniacs, destined for brilliant futures, were thwarted in their desires and succumbed to paralysis.[11] British alienists subsequently took up Bayle's themes, although they did not limit the different forms of excess so clearly to particular social classes. Neither did they place great emphasis upon physical excess - even amongst the lower echelons of society. Asylum case-notes demonstrate how ordinary doctors (whether casually or thoughtfully) assigned causes of GPI to their working and lower-middle-class charges. A typical paralytic patient appearing in the Hanwell asylum case-books between 1850 and 1880 was a tradesman whose illness had been precipitated by mental distress - business worry or business failure, marriage break-up, grief, unemployment, or debt.[12] This overall range of mental causes was not notably different for other types of the insane; however, business-related concerns were far more commonly linked with GPI. By 1880 59% of GPI cases specified business worry, ambition, or overwork as the precipitating cause of the illness, compared with less than 10% in non-GPI cases; and this stereotypical explanation for GPI contrasted with a far greater variety of physical and mental causes ascribed to other types of insanity. How alienists viewed the moral character of their patients can only be implied from these case histories; but it is noticeable that the vast majority of GPI patients during the 1860's and 1870's were described as steady, sober, and industrious.

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[11] For further discussion of the Bayle - Esquirol disagreement, see Hurn, 1994, pp 7 - 28.

Published rhetoric was more emphatic than these day-to-day asylum observations, and shows that the GPI-ambition link was extrapolated to imply the virtues of ardour, strength, and activity in the potential patient. Conolly was quick to challenge what he regarded as the prevailing French view that GPI was caused by intemperance; on the contrary, he suggested, the sufferers were usually 'rather active, moderate men'.[13] In corroboration, many of his case histories noted that the patients had 'well-formed heads'.[14] Harrington Tuke noted that the victims were not '... the ailing and the weak; but the ardent sportsman, the strong and busy worker, the men of active intellect and powerful frame ....'; this despite the fact that 'intemperate and profligate habits predispose to the disorder ...'[15] Similarly Blandford noted that GPI was usually found 'in men who are not only in their greatest vigour, but often fine, handsome, powerful men - men who have enjoyed life, and lived hard. We do not find it amongst weak, nervous valetudinarians, the subjects of hypochondria and melancholia ...'.[16] Images of the typical victim of this 'disease of manhood' bore a clear relation to dominant mid- and late-Victorian views of virility: vigour, activity, and, increasingly, physical prowess.[17] The association would remain strong throughout the history of the disease - despite changes in imagery which would

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[12] Male clinical case-books of Hanwell Asylum (1850 to 1880). GLRO, H11/HLL/B20.
[13] Connolly, 1846, p 444.
[14] Ibid, p 444.
[15] H Tuke, 1858, p 584.
[16] Blandford, 1871, p 272.
[17] The phrase is Maudsley's: Maudsley, 1868, p 411. For views of Victorian masculinity see J Tosh, 'What should Historians do with Masculinity? Reflections on Nineteenth-Century Britain', History Workshop Journal, 1994, 38, pp 179 - 202; J Oppenheim, 'Shattered Nerves': Doctors, Patients, and Depression in Victorian England (New York, Oxford: OUP, 1991), particularly Ch 5: 'Manly Nerves'; S O Rose, Limited Livelihoods: Gender and Class in Nineteenth-Century England (London: Routledge, 1992); G Bederman, Manliness and Civilisation (Chicago and London: University of Chicago Press, 1995).

appear towards and after the turn of the nineteenth century. This observation challenges the wholly negative interpretation of the general paralytic assumed by writers such as Showalter, who depict him as a favourite villain of the period whose violence and 'perversion of moral sense' were reviled.[18] Wilson's lusty portrayal, quoted at the beginning of the chapter, was an example from the era of degenerative psychiatry. In 1915, well after a link with syphilis had been made, a report published in the Lancet noted that, prior to their illness, general paralytics were generally 'efficient and productive social units of civic worth and capacity'.[19] And as late as 1928, a discussion of preventive medicine was noted to apply with great force to GPI, 'which hitherto had destroyed annually a small army of the most virile and useful section of the community, the go-ahead and active fighters in every walk of life, men who enjoyed life to the uttermost'.[20]

It is not easy to analyse how doctors might have arrived at such assumptions about disease aetiology and disease 'character'. We do not know, for example, how thoroughly alienists enquired into the previous lives of their patients (although these are sometimes recorded in great detail in case-books); nor to what extent they based their causal conclusions upon received histories or upon standard theories about insanity. The most obvious explanation for the prevailing image of GPI was its observed epidemiological pattern. Throughout its history GPI was identified far more frequently in men than in women, and at a peak age incidence of between 40 and 50

[18] Showalter, 1985, p 111; Idem, 1986, pp 88 - 110.
[19] 'Asylum reports', J Ment Sci, 1915, 61, pp 300 -301.
[20] 'General Paralysis: A Discussion which took place on Nov 1928, under the Auspices of the General Paralysis Sub-Committee at a General Meeting of the Royal Medico-Psychological Association', J Ment Sci, 1929, 75, pp 1- 30 and pp 271 - 297; on p 23.

years - a time when many men were at the heights of their careers. Much was made of this association with industrious heads of families, both in explaining why the illness came about and in emphasising its tragic waste.[21] 'Such men', noted the Edinburgh psychiatrist Robertson, ' ... occupy positions of responsibility, for as a rule those who suffer from general paralysis are no weaklings ...' The problems caused by other kinds of insanity were, he claimed, 'trivial compared with those produced by a disease such as this, which attacks the bread-winner of a family and the head of a business in the prime of his life.'[22]

The class associations of GPI were less straightforward. Whilst early observers such as Conolly, concurring with French opinion, thought that the disease affected predominantly the lower classes, later views were paradoxical. Most doctors maintained that it must be commonest amongst lower-class men, since it was seen predominantly in public asylums.[23] However, its association with the professional and upper classes - using imagery gleaned from private practice and possibly the higher classes of public asylum patient - dominated discussions until beyond the turn of the century.[24] This disjunction is not easy to explain; but although the image of masculinity presented owed much to the bourgeois Victorian ideal, admirable connotations were clearly translated to the working-classes, as the Hanwell case-notes

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[21] Tosh stresses the cultural weight attached by Victorians to the unaided male breadwinner: 'The idea that what a man did in his working life was an authentic expression of his individuality was one of the most characteristic - and enduring - features of middle-class masculinity': Tosh, 1994, p 186.

[22] G M Robertson, 'The Morison Lectures, 1913: General Paralysis of the Insane', J Ment Sci, 1913, 59, pp 185 - 221; on p 185. Although written as late as 1913, this comment was similar to views of 30 or 40 years earlier.

[23] See, for example, J Wilkie Burman, 'A Contribution to the Statistics of General Paralysis', in West Riding Lunatic Asylum Medical Reports, 1871, 1, pp 129 - 151; Mickle, 1886, p 257.

[24] Maudsley, for example, noted that it affected 'often the better classes of society': Maudsley, 1879, Vol 2, p 433.

demonstrate. This is supported by the suggestion that bourgeois images of masculinity came to apply increasingly to the lower middle and working classes as the nineteenth century progressed - particularly the qualities of pride in skilful work, financial independence and physical strength.[25] Indeed, where earlier descriptions of the GPI man stressed primarily his intellectual vigour and business acumen, later accounts also spoke of him as a 'fine specimen' or a 'strong animal' - an attribute which was easily applicable to both the middle and the working classes.[26]

To suggest that the perception of GPI was based upon epidemiological observations assumes a one-way leap from its 'natural' appearance to doctors' interpretations. But even accepting such extrapolations does not, I suggest, fully explain the prevailing imagery. The Victorian malady of neurasthenia provides an interesting comparison. Both diseases were linked to the male middle-classes, to ambition, money anxieties, overwork, and business disappointment more exclusively than were mental diseases as a whole. Despite this shared causality, psychiatrists do not appear to have made any link between the two conditions - although they noted that care must be taken not to confuse them in the early stages. Oppenheim describes how overwork - as a necessary corollary of ambition, struggle, and action - was used to differentiate male from female sufferers of neurasthenia, and thus to absolve the former of blame and retain their dignity.[27] On the other hand, the fact that sufferers had succumbed to overwork at all, she suggests, threw their moral adequacy and self-

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[25] Rose, 1992; Tosh, 1994, p 181, pp 186 - 7.
[26] George Savage noted that GPI affected men '... of good physique and active temperament ...': G Savage, Insanity and the Allied Neuroses (Cassell & Co: London, Paris, New York, 1886), p 279.
[27] Oppenheim, 1991. Sichermann too discusses how a diagnosis of neurasthenia could imply an excess of good qualities; and thus avoid the perjorative associations of other neuroses: Sichermann, 1977.

control into question. In contrast to the 'GPI man' the 'neurasthenia man' was popularly seen as highly educated, often bookish, sensitive and physically fragile - a man who all his life had been prone to nervous knocks and shocks. Although intellectually active, he was rarely cast as a specimen of vigorous manhood. Similarly, neurasthenics lent themselves far more readily to debates about creativity and insanity than did general paralytics: the latter were vigorous, hard-living entrepreneurs rather than highly-strung geniuses. Thus, although both diseases ostensibly had much in common, the general paralytic was regarded as the polar opposite of the neurasthenic: he was typically '..a strong, healthy man, in or near the prime of life, distinctly not of the 'nervous', neurotic, or neurasthenic type..'[28]

I suggest, then, that a further explanation of the imagery lay in the clinical symptoms of GPI. In Bayle's account, symptoms of ambitious monomania were clearly identified with the previous aspirations of the patient. Although later doctors acknowledged that all kinds of insanity could appear, delusions of wealth and grandeur continued to be seen as absolutely characteristic of the disease: 'The form of delusion has almost always borne reference to immense amounts of money, great power or some similar exaltation...'[29] Victorian alienists were used to extrapolating features which they observed during insanity to the previous life and character of the patient; thus such delusions were often explained as grotesque exaggerations of the general paralytic's previous personality and drive. Clouston described their content as former day-dreams which had become 'uncontrolled and uncorrected by judgement

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[28] W Osler & T McCrae (eds), The Principles and Practice of Medicine, 9e (New York and London: D Appleton & Co, 1920), p 917.
[29] Quoted in Esquirol, 1845, p 443.

and reason ... I have found that it was the vain, boastful, ambitious men before, who were the kings and millionaire general paralytics ...'[30] Maudsley noted that, in keeping with the nature of the insanity, the disease affected those of a 'genial and expansive' temperament.[31] As an extension of this, it was common to explain putative national differences in the incidence of GPI on the basis of the national character. Thus Harrington Tuke understood its florescence amongst the French to be due to 'the sanguine disposition and mercurial liveliness of the natives of our sister land ...'[32]

Just as striking as the content of the delusions was their frequently benevolent, joyful nature; a feature which gave them a certain - albeit grotesque - appeal. Even the most austere medical superintendent might find himself warming towards a patient who (as Skae described) 'offers a cheque for £75, 000 for the purchase of the asylum and promises to endow it with unbounded munificence, and to convert it into a paradise of brilliancy and bliss...'[33] This strangely appealing clinical image persisted well into the twentieth century. The American psychiatrist Southard's 1917 textbook brought out the feature graphically, as it depicted a blurred photograph of a general paralytic labelled: 'Euphoria in paretic neurosyphilis ... the head, arms and trunk were shaking with mirth, hence the indistinct outlines of the photograph ...'[34] Southard noted a description by the French psychiatrist Régis: 'The delusional generosity and

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[30] Clouston, 1875, p 197.
[31] Maudsley 1879, Vol 2, p 433.
[32] H Tuke, 1859, p 435.
[33] Skae, 1859-60, p 894.
[34] E E Southard and H C Solomon, Neurosyphilis: Modern Systematic Diagnosis and Treatment: Presented in one hundred and thirty-seven case histories (Boston: W M Leonard, Publications, 1917), plate n p 88. See also C Allbutt and H D Rolleston (eds), A System of Medicine, Vol 8 (London:

liberality of the paretic, and his willingness to lend his wealth and talents to social progress ... compare favourably with the more personal egoism of the victim of manic-depressive psychosis ...'[35] Later in the book, Southard recounted a case-history which clearly conflated the admirable character of the subject with his later decline into insanity: 'The phenomena that ushered in his last illness were mistaken by the local public for meritorious social reform measures ... indeed the public eloquence that he displayed a year before his death was quite in line with previous habits, despite the suspicious over-brilliance of language.'[36]

Observations of the disease in women appeared to follow naturally from their non-masculine characteristics. Women, it was argued, were not prone to GPI since they were not subject to the hopes and disappointments of a vigorous life. A class distinction was more clearly drawn here: the disease was held to be predominantly associated with working-class women, and exceptional amongst the better classes.[37] Although prostitutes were rarely singled out, this association was explained by placing a greater emphasis upon intemperance - naturally associated with the lower classes. Wilkie Burman, for example, who published a much-quoted statistical study of GPI, stressed the frequency of moral causes - especially brain work - in male victims. He explained its greater frequency amongst single than married women, however, on the basis that 'intemperance and irregular habits are much more common amongst single than married females ... All the single females [at Devon County

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Macmillan and Co, 1911), p 367: 'As a rule he is benevolent in his exaltation, being willing to grant power and wealth to those about him...'

[35] Ibid, p 73.

[36] Ibid, p 289.

[37] 'GPI is found very rarely with women - never, I may say, with ladies ...': Blandford, 1871, p 265.

Asylum] have been domestic servants, charwomen, or washerwomen, and in nearly all their cases there is a history of irregular or intemperate habits ...'[38] The clinical type of disease associated with females was also explicable on the basis of their previous personality: patients were predominantly seen to be depressive, quietly dementing, or displaying a 'gentler form of grandeur' - rather than noisy and expansive.[39] Such insipid clinical forms were similarly associated with less virile men, and with those who had led unexciting lives.[40] This observation is not specific to GPI: Showalter notes that in nineteenth-century psychiatry, 'even in violent dementia women were limited and bounded by the qualities of femininity'.[41] GPI, then, remained very much a 'male malady' with a vivid character which distinguished it from insanity as a whole; women general paralytics, rather than presenting a distinct disease, merged into the generality of the insane. It is quite probable that the perception of the disease in women meant that they received the diagnosis less frequently; and that this in turn strengthened the dominant masculine imagery. As a Scottish asylum physician noted: 'So many female general paralytics are, so to speak, 'colourless' cases of insanity that it leads one to think that not a few end their days in their homes or in the hospital wards of the poorhouse undiagnosed. Even in asylums these cases may not be diagnosed for some time ...'[42]

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[38] Wilkie Burman, 1871, p 133.
[39] Asylum notes bore this out: at Hanwell, the great majority of women paralytics were described as having dementia rather than mania: Female clinical case-books of Hanwell Asylum (1850 to 1900). GLRO, H11/HLL/B19.
[40] See Salomon, 1862, p 369; Clouston, 1883, p 366.
[41] Showalter, 1985, p 8.
[42] F A Elkins, 'Remarks upon Twenty-Eight Cases of Adult Female General Paralytics admitted to the Royal Edinburgh Asylum During the Five Years 1889 - 1893', Lancet, June 16, 1894, pp 1495 - 7; on p 1497.

'Guilty of Excess'

The destructive element of masculine energy - physical incontinence - was always an important aspect of the potential general paralytic's character. But exactly how this negative aspect might be incorporated into the admirable aspects of his masculinity posed alienists with a problem. Earlier accounts often spoke of moral perversion and intemperance in association with GPI; however, alienists recognised that there was a long prodrome, and were confused as to whether such traits were part of a man's previous character, or integral to the disease itself.[43] Such ambiguity often came down in favour of a man's character. Moral lapses were thus seen as a tragic contrast to his previous behaviour, rather than a culmination of it - in contrast to arguments about the ambitious and vigorous aspects of his personality. A Navy doctor observed in 1868 that there was, in some quarters, an impression that GPI was due to hard living and debauchery. He disagreed: 'As a rule, the very best men ... are those who are struck down by it, and if any of them become slaves to different vices and give evidence of insubordination, it is the result and not the cause of the disease...'[44] This argument continued to be used, particularly in relation to sexual indiscretion which was rarely claimed to be a major cause of GPI before the 1890's. Maudsley was one of the few alienists who laid much emphasis upon it; but even he thought it more likely to be an early sign of the disease: '... [The patient] breaks into sexual excesses quite foreign to his usual sober character.'[45]

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[43] See, for example, H Tuke, 1860, p 89.
[44] Mcleod, 'Naval Medical Blue Book: General Paralysis of the Insane', Lancet, Nov 19, 1870, p 723.
[45] Maudsley, 1868, p 414.

But the emphasis placed upon intemperance predictably increased during the last decades of the century, in line with trends for insanity as a whole. Historians have placed this partly in the context of a shift from moral to physical causal theories of insanity during the nineteenth century. In parallel with this was a shift from individualistic ideas of insanity to 'collective' ideas, in which madness was thought to be localised in a feared underclass of society.[46] The rise of degeneration theory provides the most compelling indicator of this change; and numerous accounts are available which concentrate variously upon the social, economic, and professional stimuli involved.[47] Figures from Hanwell asylum offer a convenient snapshot of the pattern for GPI. Towards the end of the century, interest in ambition and business anxieties declined at the expense of physical excess and heredity; in particular, the role assigned to alcoholic intemperance rose sharply during the 1890's. Far more dramatic than this was the sudden appearance of syphilis to account for a third of cases by 1900.[48] Correspondingly, during the last decades of the century a characteristic narrative of the general paralytic emerged which acknowledged the importance of his intemperance and sexuality, but which balanced these traits with their positive counterparts: after all, 'strong and vigorous men are most likely to be

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[46] See, for example, V Skultans, Madness and Morals: Ideas on Insanity in the Nineteenth Century (London and Boston: Routledge and Keegan Paul, 1975); J L Ray, 'Models of Madness in Victorian Asylum Practice', European Journal of Sociology, 1981, 22, pp 229 - 264.

[47] See for example, Dowbiggin, 1991; Jacyna, 1982; R A Nye, Crime, Madness, and Politics in Modern France: The Medical Concept of National Decline (Princeton: Princeton University Press, 1984); Idem, 'Degeneration, Neurasthenia, and the Culture of Sport in Belle Epoque France', J Contemporary History, 1982, 17, pp 51 - 68; D Pick, Faces of Degeneration: A European Disorder, c 1848 - c 1918 (Cambridge University Press, 1989); W Greenslade, Degeneration, Culture and the Novel 1880 - 1940 (Cambridge University Press, 1994).

[48] See male clinical case-books of Hanwell Asylum: 1880 (GLRO: H11/HLL/B20/15); 1890 (B20/20); 1900 (B20/28 and 29). Between 1880 and 1900 there was a decline from 59% to 22 % of cases attributed to business failure; a rise from 18% to 26% of cases attributed to intemperance; and between 1890 and 1900 a sharp rise from 2% to 38% of cases attributed to syphilis.

guilty of excess'.[49] In a neat displacement of blame, for example, George Savage deflected the sexual appetite of such a man to his typical choice of wife: 'of voluptuous physique'; the 'gross animal type of woman'.[50] Similarly, the claimed sexual allure and appetite of the (rare) general paralytic woman was often emphasised as immorality, to the exclusion of other qualities:

'It is rare indeed to come across a female general paralytic who before her illness could be described as ugly. Most of them have been well-developed and good-looking, and in this agree with the male general paralytics. Many of them are described as having been much admired by men, and as being charming wives and companions. Most of them, too, knew how to dress and make a good appearance, and ... of almost none would it be fair to say that they had been fools. But as to their morality no class of female patients ranks lower ... Where a proper history was obtained it more frequently revealed a bad or dissolute than a good life.'[51]

In a man such sexuality was regarded as part and parcel of a broad array of otherwise potentially positive characteristics. His decline was a personal tragedy: his energy and lust for life carried the seeds of his own destruction; and he brought about his own ruin at the very time when prospects were rosy. Here, the patient's life-story was mirrored by the progress of his illness - for perhaps no other disease provided such tragically graphic images of grandeur degenerating into pathos and disgust.

British alienists were reluctant, too, to place GPI within the framework of hereditary neuropathy: the assumption of previous vigour and physical health, after

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[49] Blandford 1871 p 282.
[50] Savage, 1886, p 283; p 60.
[51] Elkins, 1894, pp 1495-6.

all, did not sit easily with that of inborn infirmity. Morel's influential Traité des Dégenéréscences had cited GPI as an important indicator of rising insanity and degeneration amongst the lower classes; indeed he stressed the depth of degeneration which 'organic' insanities such as GPI and epilepsy implied.[52] Morel did not, however, regard hereditary predisposition as playing a great role in the aetiology of the disease, and his 1860 classification placed it under 'idiopathic' rather than 'hereditary' insanity.[53] During the 1870's French alienists seeking to align their specialty with Jean-Martin Charcot's school of neurology, based at the Salpêtrière, championed the hereditary nature of GPI.[54] Charcot's main interest in this context was locomotor ataxia, which he placed within a large family of 'neuroarthritides' - joint and neurological diseases regarded as having a hereditary association. The alienist Féré added GPI to the neuroarthriditic family, regarding it as an indirectly inherited insanity linked particularly to such somatic diseases as apoplexy, epilepsy, and congestion - rather than to GPI itself or to other forms of insanity.[55]

In British asylums, GPI was linked to heredity from the 1870's in a significant proportion of cases - although in lower numbers than for other forms of insanity.[56] Such figures were derived from explicit or anecdotal evidence of disease in relatives,

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[52] B A Morel, Traité des Dégenéréscences (Paris: J B Baillière, 1857), Preface, p viii; pp 345-7; as the British frequently noted, French alienists associated GPI more closely with the working-classes - particularly during the middle decades of the century.

[53] B A Morel, Traité des Maladies Mentales (Paris: Libraire Victor Masson, 1860), p 268.

[54] Golstein, 1987, esp Ch 9, pp 322 - 377.

[55] Ch Féré, La Famille Névropathique (Paris: Félix Alean, 1894). In France this view was by no means orthodoxy: the alienist Régis claimed: 'GPI abhors the neuropathic terrain, and is the most individual of cerebral diseases': quoted in G Ballet, Traité de Pathologie Mentale (Paris: Octave Doin, 1903), p 1034.

[56] At Hanwell asylum during 1880, for example, heredity was mentioned as a cause in 32% of male cases; corresponding with 45% for insanity as a whole. The figures for 1890 were 32% and 41% respectively. Male clinical case-books of Hanwell Asylum (1880, 1890).

which was routinely documented.[57] But written accounts were, as always, more dogmatic, and showed that alienists were quick to defend general paralytics against implications of degeneration.[58] Thus when Julius Mickle cautiously advised the regulation of marriage in order to prevent the propagation of GPI in the 8 - 16% of cases in which heredity was apparent, his Lancet reviewer dismissed the necessity: 'The inheritance directly from general paralysis so little affects the family that it seems a pity to attach a stigma of insanity to the offspring of the general paralytic ...'[59] The degenerationist barrister Strahan represented perhaps an extreme view when he claimed, in 1892, that familial nervous pathology was found in a fifth of all cases of GPI. Yet he acknowledged the originality of his suggestion that 'even GPI' should have careful marriage management, since it had always been regarded as the result of fast-living in healthy non-degenerates - 'indeed in some of the most finely-developed men'.[60] George Savage sympathetically placed GPI patients outside the mass of degenerates who were marked from birth for certain forms of insanity; anyone, he argued, might develop GPI - just as anyone might develop a brain tumour - but only those with neurotic inheritance could become insane in other ways.[61]

Until the turn of the century, then, most accounts of wholesale degeneration made little mention of GPI; and it was not linked to fears of the urban residuum - the

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[57] Asylum records give us some insight into how 'evidence' of hereditary insanity was important to theories of degeneration - a subject glossed over in most historical accounts. See, for example, Dowbiggin's comment that 'degeneration theory [in France] had as little relation to practice in asylums as did Freudianism ...': Dowbiggin, 1991, pp 169-70.

[58] See, for example, Skae, 1875, p 200; Maudsley, 1879, Vol 2, p 433.

[59] 'Review of General Paralysis of the Insane by W J Mickle', J Ment Sci, 1880, 26, pp 398 - 416; on p 409.

[60] S A K Strahan, Marriage and Disease: A Study of Heredity and the More Important Family Degenerations (London: Kegan Paul, Trench, Tribner & Co, 1892), p 106.

[61] Savage 1886, p 41.

diseased, corrupt, and mentally indolent classes which threatened British society. This was a disease to which anyone - indeed any doctor - might succumb. Savage noted in 1890 that the subject had become almost hackneyed, since '... as years pass on it seems to appeal to us more personally as one and another of our friends or patriots fall out of rank, victims to this malady'.[62]

The reported illness of Lord Randolph Churchill (1850 - 1895) typified the confluence of character and disease which GPI suggested - as well as the ambivalence of its portrayal at the end of the century. Churchill was a flamboyant and controversial figure whose value as a Conservative politician was constantly under debate during the Victorian period. After an inauspicious start in Parliament, he rose to lead the 'Fourth Party' to victory over Gladstone in 1885; held a number of Cabinet posts generally successfully for the following seven years; and finally ended his life as an outspoken - although increasingly frail and unpredictable - member of the opposition.[63] Churchill had always been a notoriously 'fast liver' prone to gambling, financial speculation, and womanising. He was also brilliant, energetic, hugely ambitious, and generous. During the 1880's he started to behave erratically, in ways which seemed to exaggerate his previous character: frantic bouts of activity and violent rages alternated with periods of withdrawal and apathy. At first he was diagnosed as suffering from nervous exhaustion; but by the early 1890's obvious physical signs of GPI appeared such as indistinct speech, memory loss, and fumbling. Their progression led to some painfully embarrassing performances in the House of Commons, and necessitated care by a succession of doctors including Gowers and

[62] G H Savage, 'The Warnings of General Paralysis of the Insane', BMJ, Apr 5 1890, pp 777 - 780.

Buzzard. Churchill's extravagant and outrageous behaviour worsened, soon shading into periods of delusional euphoria - later described in purple prose by his son Winston Churchill: 'In the midst of failure he is cheered by an artificial consciousness of victory. While the days are swiftly ebbing, he builds large plans for the future; and a rosy glow of sunset conceals the approach of night ...'[64] During a foreign trip in 1894, the debilitated man was taken seriously ill and returned to London in a semi-comatose state. Following a four-week decline he finally died, on 25 January 1895, of exhaustion and heart failure.

Churchill's illness was followed avidly by the general and the medical press, and reports were full of contradictory images of his pre-morbid character and diseased behaviour: 'fanciful', 'appealing', 'pitiful', 'reckless', 'ambitious folly', 'energy and audacity', 'Gallic'.[65] After his death, the Lancet presented his life, character, and illness as seamless: 'Lord Randolph Churchill's case throughout has been typical of general paralysis ... He led an over-full life in every direction ... being distinguished alike by his relentless tactics towards opponents and his irresponsible attitude towards friends, he drained to the very dregs the heady cup of politics ... while he continued to double with the life ... of the stormy statesman the life of the great nobleman and the man of pleasure, sport, and fashion.'[66] The contradictions in his character were mirrored by the tragic contrast suggested by his illness. A reporter at his last session in Parliament observed: 'So pathetic was the spectacle of his decadence that there was

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[63] R F Foster, Lord Randolph Churchill: A Political Life (Oxford: Clarendon Press, 1981).
[64] W S Churchill, Lord Randolph Churchill (London: Macmillan and Co Ltd, 1906), 2 Vols; Vol 2, p 482.
[65] The Times, 25 Jan, 1895, pp 5 - 6.
[66] 'Lord Randolph Churchill', Lancet, Jan 26, 1895, pp 239 - 240.

only one feeling of pain and regret ... It appeared impossible that the broken man, whose words now came nervously and ineffectively, could ever ... have figured as a hero of debate ... '[67] His career, his obituary concluded, was 'like that of a meteor flashing suddenly in its splendour and as suddenly quenched in the shades of night.'[68]

Churchill's was a tragic story of brilliance, dissipation, and self-ruin. Syphilis did not appear in its public telling - and this could easily be explained by the discretion of the time. Nevertheless, although the doctors who attended Churchill (certainly Gowers) must have been aware by the early '90s of possible links with venereal disease, the public narrative was entirely in keeping with contemporary medical views. By the turn of the century, however, syphilis had become far more explicitly associated with GPI. This link, the result of thirty years of speculation and negotiation, would add lurid colour to the picture of the disease which emerged at the beginning of the twentieth century.

GPI as Tertiary Syphilis

By the middle of the nineteenth century the prominent French venereologist Philippe Ricord had set the scene for a clinical and pathological explosion of syphilis. Ricord not only separated the disease from a rather vague conflation of venereal disorders including gonorrhoea and soft chancre; he also extended its boundaries by identifying three stages of disease - primary, secondary, and tertiary. 'Tertiary' lesions arose, by definition, many years after syphilis had been contracted; were found deep within the

[67] The Times, 25 Jan, 1895, p 6.
[68] Ibid, p 7.

body; could not be transmitted from mother to child; and were insidious and frequently debilitating. They affected, amongst other organs, the bones, brain, heart, liver, and muscles, and showed the typical syphilitic pathology of 'sclerosis' or 'gumma'.[69] The details of this pathology were increasingly refined by German microscopists such as Von Baerensprung and Virchow, allowing a fairly satisfactory consensus on post-mortem diagnosis.[70] Complex negotiations, however, surrounded the problem of how a variety of symptoms during life might be assigned to the disease. Syphilis was notorious for imitating practically any other form of disorder, leaving it apparently little identity of its own: '... All the various phenomena of disease due to syphilis are imitations of other, non-specific type forms ... We have absolutely no malady which is peculiar to syphilis ...'[71] This sinister ability gave it a reputation for being the most dissembling of diseases - a wolf in sheep's clothing. Ricord's warning was often repeated: 'La vérole vieille a la mine honnête'.[72] In the face of such clinical nebulousness, many doctors resorted to the assumption that response to anti-syphilitic treatment was all that could define syphilis during life; an ancient concept which Fleck described as the 'empirical therapeutic' definition of the disease.[73]

'Cerebral syphilis' - a manifestation of tertiary lesions in the brain or meninges - was described increasingly frequently after Ricord. The usual result was local

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[69] P Ricord, Traité Pratique des Maladies Vénériennes, ou Récherches critiques et experimentales sur l'inoculation appliqué a l'étude de ses maladies ... (Paris: J Rouvier et E Le Bouvier, 1838), p 136; for a later account of tertiary syphilis, see A Fournier, Leçons sur la Syphilis Tertiaire (Paris: Bureau du Journal l'École de Médecine, 1875).
[70] Crissey and Parish, 1981, p 219.
[71] J Hutchinson, Syphilis (London, Paris, New York: Cassell & Co, 1887), p 485.
[72] Quoted in A Fournier, La Syphilis du Cerveau (Paris: G Masson, 1879), p 8; see also Hutchinson, 1887, p 485 on syphilitic simulations.

paralysis or hemiplegia, cranial nerve lesions, or convulsions - although assigning such symptoms to syphilis was always problematical.[74] In addition, there seemed to be no doubt that such lesions could manifest themselves as insanity; they could, after all, appear as practically any kind of neurological syndrome, in keeping with their dissembling nature. Such insanity was termed 'symptomatic insanity' in the British literature, since it was a direct symptom of a demonstrable syphilitic lesion. Whether clinical features of GPI could arise from such a cause was less obvious; but the earliest work suggesting a link between the two diseases made this assumption. Esmarch and Jessen, routinely cited as the first to discover the syphilitic aetiology of GPI, thus suggested in 1857 that some cases of general paralysis might be a manifestation of tertiary syphilis.[75] Although they assumed that GPI lay within the framework of tertiary syphilis, the German researchers began to shift the emphasis of causal evidence in a way which would be extremely important. Rather than stressing pathological identity in making the link, they turned to simple statistics: the proportion of general paralytics with a history of syphilis. Furthermore, they challenged the therapeutic definition of syphilis: just because such cases of GPI did not respond to mercury, they claimed, did not mean that they were not syphilitic in origin.[76] Following Esmarch and Jessen, alienists in Scandinavia seized upon the

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[73] Fleck, 1935.

[74] See, for example, H Thompson, 'On Paralysis arising from Syphilitic Affection of the Brain', Lancet, 1857, I, p 377; J B Chapin, 'Cases illustrating the pathology of mental disease arising from syphilis infection', American Journal of Insanity, 1858-9, 15, p 249.

[75] F Esmarch and W Jessen, 'Syphilis und Geistesstörung', Allg Ztschr f Psychiat, 1857, 14, p 20, transl in M Moore & H C Solomon, 'Contributions of Haslam, Bayle, and Esmarch and Jessen to the History of Neurosyphilis', Archives of Neurology and Psychiatry, 1934, 82, pp 830 - 839. Quetel, for example, writes: 'In 1857 ... Esmarch and Jessen affirmed that syphilis was the cause of general paralysis of the insane ...': Quetel, 1990, p 162.

[76] For a fuller discussion of this work see Hurn, 1994, pp 47 - 49.

link, and many numerical studies were published during the 1860's and '70's which examined the proportion of general paralytics with a history of syphilis.[77] Such studies, clearly shifting attention from pathology to clinical statistics, gave variable results; but many indicated, startlingly, that syphilitic antecedents were found in between 80 and 90% of sufferers.

The reason for this surge of interest in Denmark, Finland and Sweden is a fascinating question beyond the scope of my study; but from a British point of view the Scandinavian claims were taken up with only slight interest until the 1880's. One ostensible reason was the problem of participation in research. Large-scale studies in Scandinavia, British doctors claimed, were relatively easy to carry out since patient numbers were smaller, and asylums were centralised allowing more efficient follow-up of cases. In contrast British asylums appeared to prohibit any form of organised research, and record continuity between hospitals, out-patient clinics and asylums was extremely poor. There were more general obstacles too: insane patients could not offer reliable information about their medical pasts, and the knowledge of relatives was often inadequate or reluctantly given. Even patients with their mental faculties intact might have unrecognised disease, or might hide such a shameful history; and in women these problems of concealment were compounded.[78]

Possibly more important than these practical difficulties was the major conceptual leap required to make a GPI-syphilis link on the basis of numbers. Simple statistical research had been carried out in a variety of fields of medicine during the

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[77] For overviews see Griesinger, 1867, p 192; E Lancereaux, A Treatise on Syphilis, Historical and Practical , 2 vols (London: New Sydenham Society, 1868), p 64.

nineteenth century, but the validity of probability-based medical knowledge was constantly debated; and in the realm of syphilis and insanity, numerical enquiry was obviously full of potential flaws.[79] The traditional pathological or therapeutic definitions of syphilis dictated that GPI could not be accepted as part of the disease, even in those cases where a history of both was established: the post-mortem lesion of GPI was not 'typically' syphilitic; and the insanity did not respond to mercury.[80] In order to entertain the link, doctors had to accept that these traditional criteria were obsolete, that syphilis could affect the individual body in more elusive and pervasive ways than were manifested by gummata, and that statistics was a valid method of tracking this reformulation of the syphilitic domain. These steps were made possible by a sweeping reconceptualisation of syphilis during the last two decades of the century, championed by the flamboyant French 'syphilographe' Alfred Fournier.

Fournier and Venereology: Domains of Syphilis

The specialty of venereology had a strong tradition in France, and during the first half of the nineteenth century a number of respected schools were established around teachers such as Alibert at St Louis, Ricord at l'Hôpital du Midi, and Diday at

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[78] In the French context, see J Harsin, 'Syphilis, Wives, and Physicians: Medical Ethics and the Family in Late Nineteenth-Century France', French Historical Studies, 1989, 16: 1, pp 72 - 95.

[79] There are few coherent accounts of the place of statistical enquiry in nineteenth century medicine, but see: T M Porter, The Rise of Statistical Thinking (Princeton: Princeton University Press, 1986); S Peller, Quantitative Research in Human Biology and Medicine (Bristol: John Wright & Sons Ltd, 1967); J R Matthews, Quantification and the Quest for Medical Certainty (Princeton: Princeton University Press, 1995).

[80] See Gower's statement of these objections: W R Gowers, 'Syphilis and the Nervous System' in B Hill and A Cooper, Syphilis and Local Contagious Disorders (London: Smith, Elder & Co, 1881), pp 204-56; on p 225. Gowers, however, conceded that occasionally tertiary syphilis produced GPI-like symptoms.

Lyons.[81] Despite this precedent, it was only through prolonged struggles that 'syphilographes' achieved the recognition they desired. Until well into the twentieth century the specialty was intimately connected with, and in many ways subordinated to, that of dermatology: thus the first chair of syphilology, instituted in 1880, was combined with that of skin diseases, and even then the Paris Faculty agreed to recognise it only reluctantly, since it considered venereology of dubious scientific interest.[82] This disinterest was mirrored by what many doctors perceived as apathy towards public health measures against syphilis for much of the nineteenth century. During the last quarter of the century, however, venereologists managed to raise their profile considerably: specialisation on the German model was regarded as increasingly desirable by the French authorities; and increasingly the problem of syphilis as a social scourge was recognised. In the rise of French venereology, Alfred Fournier - former pupil of Ricord - was widely acknowledged as master and champion from the 1860's onwards; and his name was particularly associated with moves towards a more rigorous professional structure, organised and compulsory education for medical students, and public health measures against syphilis.[83] In 1871 he implemented the first Cours Clinique Complementaire in Venereology at the

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[81] According to Quetel, the term 'syphilographie'was first coined in 1842; see Quetel, 1990, p 136, note 8.

[82] See G Weisz, 'The Development of Medical Specialisation in Nineteenth-Century Paris', in A La Berge & M Feingold (eds), French Medical Culture in the Nineteenth Century (Amsterdam: Editions Rodopi, 1994), pp 149 - 188; on p 171. Historians also tend to see the latter part of the century as one of stagnation in terms of research into syphilis; see, for example, Crissey and Parish, 1981, p 230: 'The late century literature on syphilis was over-ripe and repititious ...' The first French journal devoted to syphilis - La Syphilis - appeared in 1903, in clear response to a sense of apathy in the field: Quetel, 1990, pp 137 - 138.

[83] According to Quetel, Fournier made 'syphilis a respectable branch of medicine ...': Quetel, 1990, p 136; according to Alain Cobain , Fournier 'was the first syphilographer of ... all times', quoted in Quetel, p 136. See also M A Waugh, 'Alfred Fournier, 1832 - 1914: His Influence on Venereology',

Lourcine Hospital; by 1876 he had taken this to the St Louis Hospital; in 1880 he took the first chair in Diseases of the Skin and Venereal Disease at the Academy, and simultaneously implemented the first official course on venereal diseases at both hospitals. His concern for the social consequences of syphilis stimulated the foundation of the 'Société Francais de Prophylaxie Sanitaire and Morale' in 1899, and led him to participate in numerous national and international efforts against the disease until well into the twentieth century.

Fournier continually reiterated two concerns: that syphilis represented an ever-increasing threat to society; and that energetic prophylactic measures should therefore be taken against it. Furthermore, he explicitly mirrored its invasion of society with its perceived appropriation of the individual body. Such rhetoric drew upon a venerable tradition in nineteenth-century syphilis literature: work upon tertiary syphilis had provided ample evidence that it was no respecter of any part of the body; it was 'insidious', 'deceitful', the 'unrelenting creditor'. As the century progressed, more organs were added to the list of those affected by the tertiary disease, and Fournier's ominous pronouncement in 1887 was typical: 'The more we have studied syphilis in its clinical and anatomical aspects, the more we have seen the limits of its pathological domain unfold through a series of unforeseen conquests ...'[84] To Fournier, syphilis inextricably linked pathology, specialty, and society; and its domain, he insisted, was clearly outstripping traditional perceptions of its definition

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Br J Venereal Diseases, 1974, 50, pp 232 - 236; H Gongerot et L Brodier, L'Hôpital Saint-Louis et la Clinique d'Alfred Fournier (Paris: J Peyronnet & Cie, Éditeurs, 1932).

[84] A Fournier, La Prophylaxie Publique de la Syphilis (Paris: J-B Baillière et fils, 1887), p 7. In 1875, the Army surgeon Welch added the aorta to the list of targets - a link which relied upon microscopic

and nature. His work upon locomotor ataxia, De L'ataxie locomotrice d'origine syphilitique (1876), introduced a number of ideas which would be integral to his project of expansion over the following thirty years.[85] Contrary to current opinion, he claimed, syphilis was an important cause of locomotor ataxia, presenting no differently from those cases due to other causes. One by one he countered the narrow and 'detestable' objections to this link by denying the traditional definition of syphilis and posing new definitions.[86] Locomotor ataxia - claimed the traditionalists - manifested itself with no peculiar syphilitic symptoms or lesions; was not influenced by anti-syphilitic treatment; and revealed a history of syphilis only by chance. Syphilis - countered Fournier - could no longer be characterised by distinctive pathology; and treatment might well be ineffectual in an irreversible pathological process. His statistical studies revealed that, of 30 patients with locomotor ataxia, 24 had antecedent syphilis; it was common sense, he urged, that these figures were too significant to be explained by chance alone. In conclusion, Fournier invoked the image of syphilis invading new, hitherto unimagined domains: why should this disease, which respected no part of the body, not also invade the posterior columns of the spine?[87]

Three years later, in La Syphilis du Cerveau (1879), Fournier turned his attention to GPI.[88] The ideas presented here are in one sense surprising: Fournier hesitated to suggest a firm link between GPI and syphilis since he considered the

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pathological criteria of syphilis: F H Welch, 'On Aortic Aneurysm in the Army and Conditions Associated with it', Med-Chir Trans, 1876, 41, pp 59 - 77.

[85] A Fournier, De L'ataxie locomotrice d'origine syphilitique (Paris: G Masson, 1876).

[86] Ibid, p 15.

[87] Ibid, p 28.

figures of antecedents unconvincing. However, his main argument was to challenge the traditional view that cerebral tertiary syphilis might manifest itself as true GPI. The cases which Scandinavian studies linked to syphilis were, he claimed, nothing more than good imitations of GPI by tertiary syphilis - 'pseudo-general paralysis of syphilitic origin'[89]. This imitation was easily revealed as merely syphilis rather than true GPI, since it was curable by anti-specifics. This model - in which true GPI was opposed to 'syphilitic pseudo-GPI' - became known as the 'duality' theory. In separating GPI from the realms of direct syphilitic pathology, however, Fournier did not exclude the possibility of some connection between the two. He introduced a second model in which syphilis produced effects apart from its tangible pathological lesions, through a 'general perturbing influence' on the organism.[90] This indirect model of influence - sometimes called the syphilitic 'diathesis' - was certainly not new: it had long been claimed that syphilis might cause a general lowering of the economy, rendering a patient vulnerable to other diseases; or that it might prepare the cerebral soil for other morbid processes.[91] Over the following fourteen years, however, Fournier would develop this vaguer model of syphilitic action into his new conception of the realm of syphilitic disease. He would also change his mind dramatically about the statistical evidence in GPI patients - and, like a true convert, he would be evangelical about his change of heart. Meanwhile, whilst his general

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[88] Fournier, 1879.
[89] Ibid, pp 337 - 8.
[90] Ibid, p 340.
[91] Such rather vague descriptions were in a completely different frame of reference to that of tertiary cerebral syphilis. For a fuller discussion, see Hurn, 1994, pp 37 - 46.

syphilitic campaign continued through the 1880's, his duality theory gave him - ironically - the reputation for being antagonistic to the GPI-syphilis link.

Fournier turned his attention next to the domain of hereditary syphilis; and it was this aspect of his work which provided the most important groundwork for the acceptance of the GPI link during the following decades. The knowledge that syphilis could be transmitted to the infant causing still-birth, sickliness and deformity was almost as old as the history of syphilis itself. During the nineteenth century the phenomenon was documented more carefully, and Diday's 1854 Treatise on Syphilis of the New-born and Infants at the Breast offered a classic description of congenital syphilis which provided the basis for later refinements.[92] Whilst Diday and his French successors continued to locate the initial manifestations of the disease in the first few months of life, the influential London Hospital surgeon Jonathan Hutchinson suggested that doctors should extend their scrutiny to many years after birth.[93] From 1858 onwards he described the phenomenon of 'late congenital syphilis', in which children and young adults betrayed their diseased heritage through a number of features - classically notched teeth, chronic interstitial keratitis, and nerve deafness.[94] These manifestations, which might appear up to thirty-five years after an apparently cured neonatal disease were, according to Hutchinson, 'amongst the marvels of pathology'.[95] Nevertheless, his style for the next fifty years remained that of the

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[92] P Diday, A Treatise on Syphilis in New-Born Children and Infants at the Breast, transl G Whitley (London: New Sydenham Society, 1859).
[93] See H Hutchinson, Jonathan Hutchinson: Life and Letters (London: WM Heinemann Medical Books Ltd, 1946); H Ellis, 'Jonathan Hutchinson (1828 - 1913), J Med Biography, 1993, 1:1, pp 11 - 16.
[94] J Hutchinson, 'On the means of recognising the subjects of inherited syphilis in adult life', Med Times Gaz, Sept 11, 1858, pp 264 - 265.
[95] J Hutchinson, 'Notes on Syphilis: Address to the Pathological Society; and Discussion', Lancet, Feb, 1876, pp 201 - 206; 534 - 540; on p 204.

careful, moderate empiricist: 'Mr Hutchinson', reported the Lancet in 1863, 'has unveiled the nature of a large and most important class of infantile diseases ... [Nevertheless] he is not an alarmist ... He does not re-echo the vague declamation of those unclean transcendentalists in medicine who review the whole army of sufferers from constitutional disease with the cry of 'Hereditary Syphilis!' and would have us believe that the whole world is affected with the malady'.[96]

This comment reflects a typically conservative British response during the 1860's and '70's; and it is probable that the targets of the attack were French degenerationists.[97] Alain Corbin, however, suggests that even in France syphilis attracted little interest amongst degenerationists before the 1880's.[98] It fell to Fournier and his school to promote a far more wide-reaching and disturbing concept of the disease, expressed in publications such as Syphilis et Mariage (1881) and La Syphilis Héréditaire Tardive (1886).[99] Through these works, Fournier attributed a whole host of vague but profound neurological, intellectual and moral afflictions to the 'heredo-syphilitic' - afflictions which haunted the patient throughout his adult life. To explain these, Fournier drew upon his concept of the syphilitic diathesis: ' ... The syphilitic influence of parents does not show itself only in their children by ordinary symptoms of syphilis, but also by morbid conditions and morbid dispositions, which have nothing syphilitic in themselves, and which exhibit no external evidence agreeing with the classical symptomatology of the disease, which are even as different

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[96] Review of J Hutchinson, A Clinical Memoir on Certain Diseases of the Eye and Ear, consequent on Inherited Syphilis (London: Churchill, 1863), in Lancet, Aug 22, 1863, pp 222 - 3.
[97] See Pick, 1989, p 179 onwards.
[98] Corbin attributes this to a belief that syphilis was a dying disease - a suggestion which is difficult to reconcile with accumulating work on tertiary syphilis: Corbin, 1995.

as possible therefrom, but which do not any the less constitute modified expressions of the ancestral diathesis ... the indirect legacy of syphilis.'[100] In France a rich literary seam arose from this portrayal: the 'heredo' would come to represent, during the early twentieth century, the epitome of decadence - a sickly, hypersensitive, half-mad aesthete.[101] Corbin's study offers a vivid picture of the venereologist's charismatic construction of this concept, and its promotion within popular culture as the embodiment of the degenerative taint. Central to his thesis is the imperialism of Fournier and his fellow venereologists: by constructing hereditary syphilis as inexorable, pervasive, and (most importantly) non-specific in its manifestations, the school, he suggests, both exploited and fuelled contemporary bourgeois fears about the threat of unbridled sexuality.

The hereditary threat was at the heart of Fournier's multifarious projects from the 1880's onwards. In 1887 he published the results of an enquiry commissioned by the Academy of Medicine, La Prophylaxie Publique de la Syphilis.[102] The work laid out in vivid detail the terrible social consequences of syphilis, including infant mortality, incapacity to work, invalidity from the army, disastrous marriages, and sterilisation. On its broadest scale, the work articulated the widespread fear that syphilis, together with a swathe of other organic diseases, was causing depopulation and degeneration of the race. These threats demanded energetic defensive measures, and Fournier clearly linked their implementation to the creation of venereology as an

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[99] A Fournier, Syphilis and Marriage, transl A Lingard (London: David Bogue, 1881); Idem, La Syphilis Héréditaire Tardive (Paris: Masson, 1886).
[100] Fournier, 1881, p 57.
[101] Corbin, 1995, p 121; See also Quetel, 1990, pp 165 - 175.
[102] Fournier, La Prophylaxie Publique ... , 1887.

organised discipline. Many of his recommendations were laid out in the Prophylaxie; however, although the Academy officially approved them, Fournier was still bemoaning the delay in their implementation at the beginning of the twentieth century. There should be, he urged, a more sweeping medical surveillance of the army and navy. The regulation of prostitutes with venereal disease should be transferred from a police function to an exclusively medical function. New hospitals specialising in venereal disease should be opened in each town, with high quality outpatient clinics attached. No longer should consultations be 'unobliging, inconvenient, humiliating, odious'; the new dispensaries would open at times convenient to the patient, and would offer private consultations by specialists.[103] Most urgently, medical education should be transformed by opening up the venereal sections of hospitals to students. The lock hospital for prostitutes at St Lazare, currently 'a tomb of wasted pathological and clinical material', should be transformed into a great school of syphilis such as the Lourcine; no longer a closed penitentiary, it would become a tolerant institution of education. Fournier's pleas sprang from a central conviction: that syphilis should be transferred from the sphere of the moral to the sphere of the medical, in a way which fruitfully aided the establishment of venereology as a specialty.[104] Only these reforms would result in the creation of a 'scientific movement with regard to syphilis.'[105]

In Britain, traditional moderation meant that there was a vocal strand of opposition to Fournier's doctrines. Hutchinson continued to urge against hysteria

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[103] Ibid, p 35.

[104] This is not to say that Fournier's own code of morality did not permeate his writings: see, for example, Harsin, 1989.

when discussing congenital syphilis: he insisted that neurological and mental manifestations were neither common nor profound, and that there was no tendency to degeneration in syphilitic families. Idiocy and insanity, he stressed, were rare; in fact congenital syphilitics showed the whole range of mental ability as in any other sample of the population.[106] In 1898 he still affirmed that ' ... there was very little bad health in the general population which could be attributed to hereditary syphilis ... the popular fear of the hereditary disease was very much exaggerated...'[107] One concrete objection was a recognition that syphilis could only be transmitted over two generations; Fournier, in contrast, tentatively espoused third-generation transmission (that is, the transmission of the disease by congenital syphilitics to their off-spring), which clearly offered greater rein for degenerative effects.[108] But Hutchinson was by now an ageing surgeon, and his views cannot be taken as necessarily representative of the medical profession. It is certainly probable that British doctors embraced the concept of pervasive degeneration less enthusiastically than did their French counter-parts.[109] Writers such as Strahan - a barrister who provided propagandist images of the dangers of syphilis at the end of the century - represented the extreme of opinion. Whilst acknowledging that the disease was not truly hereditary, he affirmed its supreme importance in race deterioration; first, since it exploited weak degenerate organisms; and second, since it lowered resistance, allowing degeneration to take

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[105] Fournier, La Prophylaxie Publique ..., 1887, p 19.
[106] Hutchinson, 1876, p 204; Idem, 'Lettsonian Lectures on some moot points in the natural history of syphilis' (Address to the Medical Society of London), BMJ, Feb 6, 1886, pp 239 - 242; pp 279 - 282.
[107] Idem, 'A Discussion on Some Aspects of Congenital Syphilis', BMJ, Oct 15, 1898, pp 1149 - 1155; on pp 1149 - 50.
[108] Debates about the mode of transmission - and the relative roles of male and female parent - were extremely complex. For further discussion, see Lomax, 1979; Corbin, 1995; Spongberg, 1998.

over: 'Aided by drunkenness, poverty and squalor, syphilis is largely responsible for that residuum of humanity to be found in the dark places of our great centres of population, from which are recruited the consumptive, the scrofulous, the epileptic, the prostitute, the idiot, the habitual drunkard, the instinctive criminal, and the insane.'[110] Strahan's lurid description of the syphilitic child drew attention to the visible marks of degeneration, and to a metaphorical loss of innocence which the sexual sins of its parent had caused: 'All syphilitic children are ill-developed, miserable, puny things ... Their little faces are withered, pale, and pinched; their noses become flat, their heads are large ... their cheeks seared with the scars of old sores; and over all there is a strange uncanny look of age and suffering which is repulsive, and strangely at variance with the cherub-like features and innocence of the healthy infant.'[111]

Strahan's account demonstrated well that the degenerationist concept was fluid enough to by-pass problems of strict heredity, as the distinction between inherited and acquired factors was frequently blurred.[112] It also illustrates that - for some observers at least - Spongberg's contention is accurate: '... the syphilitic body was set to become the quintessential degenerate's body'.[113] Whilst most British doctors were suspicious of such sensationalism, many accepted the breadth of mental and neurological dangers which congenital syphilis carried, and increasingly spoke of the disease in degenerationist terms. In 1895, the Lock surgeon Cooper noted that

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[109] See Pick, 1989; Lomax, 1979; and for a later period R Soloway, 'Counting the Degenerates: The Statistics of Race Deterioration in Edwardian England', J Contemp Hist, 1982, 17, pp 137 - 164.
[110] Strahan, 1892, p 143.
[111] Ibid, p 152.
[112] See Nye, 1982.

defective development of the brain in congenital syphilis commonly caused ' ... congenital idiocy ... dullness and apathy, want of mental control ... inability to concentrate the mind and feeble-mindedness ...'[114] By the early twentieth century, Marshall felt justified in devoting a whole chapter of his influential venereology text to heredo-syphilis of the nervous system. Careful study, he predicted 'would probably reveal syphilis in most idiots ... Syphilis is the hereditary disease par excellence. Its effects are more inevitable, more multiple, more diverse, and more disastrous in their results on the progeny and the race than in the case of any other disease.'[115]

'Parasyphilis' and the Power of Statistics

By the 1890's, then, Fournier had paved the way for a new understanding of the social and clinical march of syphilis. Whilst hugely enthusiastic about the power of statistical enquiry, he had remained unconvinced for many years that there was a good numerical link between GPI and syphilis - indeed he was regarded as leading the 'traditionalist' school against the Scandinavians.[116] But in 1894 he published a typically dramatic and polemical work which revised his earlier theories: Les Affections Parasyphilitiques.[117] Syphilis, he claimed, could indeed cause true GPI, not merely the imitation 'pseudo-GPI'; moreover, although other factors might play a part, syphilis was its most active cause. The term 'parasyphilis' marked a further step

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[113] Spongberg, 1998, p 239.
[114] A Cooper, Syphilis, 2e, ed E Cotterell (London: J&A Churchill, 1895), p 397.
[115] C F Marshall, Syphilology and Venereal Disease (London: Bailliére, Tindall, & Cox, 1906), p 317.
[116] See D E Jacobson, 'The Relationship between GPI and Syphilis', J Ment Sci, 1892, 38, pp 175 - 185.
[117] A Fournier, Les Affections Parasyphilitiques (Paris: Rueff et Cie, Éditeurs, 1894).

in the reformulation of venereal disease, again expanding the concept of diathesis which Fournier had exploited in his account of hereditary disease. It referred to a group of conditions (of which GPI and tabes dorsalis were the most important) which were syphilitic in cause or provenance, but not in nature. This causal relationship between two diseases of different clinico-pathological nature finally laid to rest, Fournier claimed, the two accepted criteria of syphilitic identity: 'All that is syphilitic can be defined by its pathological lesions'; and: 'All that is syphilitic can be cured by anti-syphilitics'.[118] These tenets, he wrote, were now out-moded; indeed he proclaimed his ideas as evidence that the pathological era in medicine was obsolete: 'This framework [pathology] was well able, in the past, to shape all the scientific evidence of an epoch - but now, through scientific progress, it must be abandoned ... We are precisely at that point ...'[119]

The book instead presented statistical enquiry as the supreme test of causal relationship - well qualified to over-ride earlier definitions of the boundaries of syphilis. Although published statistical studies gave a range of results, 'triaging' them, Fournier claimed, enabled him to select the most accurate: these, together with his own studies, revealed that 66 to 80% of general paralytics had contracted syphilis in the past. Fournier was first and foremost a clinician; he treated hypothesising with disdain, and was notoriously uninterested in laboratory medicine. To him the observation of statistical evidence alone could lead doctors to scientific truth.[120] He

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[118] Ibid, pp 186 - 192.
[119] Ibid, p 193.
[120] Fournier had used similar rhetoric in an earlier study on the distribution of syphilis in women, using figures to refute the idea that the disease was confined to the underclasses: 'So you see, gentlemen, the significance of such a statistic? ... What a ... crushing reply, to those who would have us believe that

related with relish how, during the past fifteen years, he had opened his eyes and learned to appreciate the compelling power of numbers. In assessing his theory, the reader must simply accept or reject the bare figures: '... to refuse to admit this connection ... is nothing less than to close one's eyes to the light, and to ignore ... what I'd dare to call conclusive clinical evidence.'[121]

Fournier's book was full of vivid, persuasive language which insisted that he was leading readers to the light of reason in forging the link between syphilis and general paralysis. This was central to his dramatic style, and he clearly over-stated his radicalism with regard to statistical enquiry: such studies had been published frequently over the previous thirty years. More important was his statement of a new definition of syphilis: a further powerful symbol of the disease's expanding domain, and of his own specialty's central rôle in public health. Parasyphilis, he announced, finally demonstrated the true nature of syphilis: ' ... More loaded with grave consequences, more formidable, menacing, dangerous and poisonous than it was supposed until now ...'[122] A central defining feature of the parasyphilitic diseases - their resistance to anti-syphilitic treatment - made prophylactic measures absolutely central to control: 'Since therapeutics ... have left us undefended, what [but prophylaxis] will be our safeguard against these terrible derivatives of syphilis....?'[123] Little had changed in the profession, he pointed out, since his Prophylaxie of 1887: syphilis was still poorly understood amongst doctors; only a small number gained

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syphilis is the monopoly of the demi-monde!': A Fournier, 'Document statistique sur les sources de la syphilis chez la femme ...', Bull de l'Academie de Médecine, 25 Oct 1887, quoted in Quetel 1990, p 137

[121] Fournier, 1894, p 183.

[122] Ibid, p 203.

experience by attaching themselves to special hospitals; the subject was rarely tested in examinations. His discovery, he hoped, would finally change this indifference: 'Perhaps now our faculty will understand that there is a public interest ... in giving our future doctors a sufficient education about one of the most frequent and grave illnesses that they will treat ...'[124] His final appeal to 'colleagues, hygienists and government' vividly highlighted the implications of the new link: 'Beware! Truly syphilis is a more redoubtable enemy than you think. She doesn't stick to minor annoyances ... she carries other dangers more serious and menacing yet ... nothing less than general paralysis and locomotor ataxia ...'[125]

Fournier was well aware that his extension of the domain of syphilis and, by implication, the specialty of venereology, might be perceived as a threat by doctors in other fields of medicine. He used this to great dramatic effect in Les Affections Parasyphilitiques. Twenty years ago, he remarked, it was a brave doctor that was audacious enough to suggest a connection between GPI and syphilis; indeed earlier in his career even he did not dare to believe in the link: 'I thrust away such a doctrine as I would a heresy ... so much so that, to interpret observations I imagined a new morbid type - pseudo-general paralysis of syphilitic origin'.[126] Even now, his ideas were outside those of official science; they were 'judged "subversive" and "revolutionary" by certain alienists, troubled in the calm of their asylums.'[127] Fournier conceded that nothing was more natural than the antagonism of these

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[123] Ibid, p 359.
[124] Ibid, p 362.
[125] Ibid, p 365.
[126] Ibid, p 166.
[127] Ibid, p 163.

colleagues who saw the continual encroachment of syphilis upon various domains of pathology. General paralysis 'was considered as a sacrosanct morbid entity, absolutely autonomous, rigorously independent of all affiliation, not in the least susceptible to servitude to another illness ...'[128]

How much of this was accurate, and how much dramatic rhetoric, is not clear. It is quite possible that Fournier exaggerated opposition in order to highlight the transformation from ostensible heresy to 'classic truth'; in particular, it seems unlikely that he would have been cowed by opposition during the 1880's, and he usually claimed that the persuasiveness of statistical evidence was the main reason for his volte-face on GPI. The French historian Quetel goes beyond Fournier's own explanation, suggesting that he cleverly softened his psychiatric antagonists by retaining the term 'pseudo-GPI', before presenting his more radical ideas. This retrospective explanation of motives retains the image of Fournier as heroic and consistent![129] But it is readily accepted that Fournier was often seen to be trespassing upon the domain of other specialists, and that this caused some resistance to his doctrines.[130] He had a reputation for attributing a great number of symptoms to syphilis which colleagues regarded with scepticism: the assumption that the more doctors looked the more they saw syphilis was a common charge - particularly as the clinical criteria could be interpreted widely.[131] Fournier defended himself with good

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[128] Ibid, p 184.
[129] Quetel, 1990, p 163.
[130] See J Darier, 'Alfred Fournier 1832 - 1914', Extrait des Annales de Dermatologie et de syphiligraphe, Juillet, 1915, pp 519 - 520; Gongerot et Brodier, 1932, p 113; Waugh, 1974, p 235; Crissey and Parish, p 223; Quetel, 1990, p 163: 'For several decades, Fournier and his successors ... had to contend with general incredulity.'
[131] See W R Gowers, A Manual of Diseases of the Nervous System (London: Churchill, 1886 - 8), p 289: '[Fournier's] statistics were received with doubt, because syphilitic patients constituted his field

humour: "'M'accuser de voir la syphilis partout", souriait-il, "moi qui passe mes journées à rétorquer l'erreur et à dire aux malades: Mais non mon bon ami, vous n'avez pas la syphilis!'"[132] It was part of his style to abhor such inter-specialty antagonism; he constantly urged that all doctors should work together, and sometimes claimed that alienists were unwilling to pull their weight.[133] But even his own eulogists believed that there was some justification in the accusations.[134]

Charcot and his school were often-quoted opponents of his doctrines, particularly in relation to tabes dorsalis.[135] To Charcot, clinical disagreements were closely allied to territorial concerns, and he was reputed to have disseminated the idea, through his pupils, that Fournier was over-stepping the boundary into neurology. Yet he usually confined his criticisms to Fournier's theory itself. In 1887, he used the forum of one of his Tuesday Lessons to refute the syphilitic provenance of tabes. Statistics, he admitted, revealed a high proportion of syphilitic antecedents in tabetics: '... After all', he remarked dryly, 'who can argue with numbers?'[136] But he remained wedded to the traditional concept of syphilis as defined by pathology and response to treatment; since these criteria did not apply in the case of tabes, he argued, it could

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for observation ...'; W H B Stoddart, 'General Paralysis and Syphilis: A Critical Digest', J Ment Sci, 1901, 47, pp 441 - 458; esp pp 445-6.

[132] Quoted in Waugh, 1974, p 235.

[133] He expressed ironic astonishment that the alienist Régis had called for prophylactic measures against syphilis in response to the rising tide of GPI: '...Who would have bargained for alienists becoming involved in this affair? Who would have expected to see them become our collaborators in the study of the origin and means of repression of syphilis?': Fournier, 1894, p 166; see also p 359.

[134] See Darier, 1915, p 519.

[135] 'Even the illustrious Charcot', wrote Débove delicately in his 1917 eulogy of Fournier, 'was indignant at the suggestion of the syphilis-tabes link ...': M G M Débove, A Fournier: Éloge prononcé à l'académie de médecine, dans la séance annuelle du 11 décembre 1917 (Paris: Masson et Cie, Éditeurs, 1917); Harsin, 1989.

[136] Charcot the Clinician: The Tuesday Lessons, Excerpts from Nine Case Presentations, 1887 - 88 by J-M Charcot, transl with commentary by C G Goetz (New York: Raven Press, 1987), Ch 1: 'Syphilis,

not be a syphilitic disease.[137] To Charcot, who regarded tabes as a hereditary disease belonging to a stock of neuro-arthritidic conditions, syphilis was a red herring which diverted attention from familial influences. Ironically, it was these influences which, he claimed, patients and families would try to conceal, rather than a history of syphilis! [138]

This territorial aspect of Fournier's work has led Alain Corbin to take a constructivist view of his scientific theories: he suggests that the venereologist 'created from nothing the notion of parasyphilis, a first attempt to extend the domain of the disease beyond its specific manifestations.'[139] Such a claim is clearly extreme. It ignores the accumulation of statistical and other evidence with which both Fournier and his predecessors were grappling. It is, I suggest, simplistic to claim that such a causal theory might arise purely from ambitions of expansion; an equally rational alternative is that causal convictions led to a perceived need for specialty expansion. Notwithstanding this, Fournier did create a revolution in the perception of the domain of syphilis which would have a profound effect upon the work of British alienists.

Accepting the Link in Britain: 1880 - 1910

During the 1880's, British doctors were still resistant to the possibility of the GPI-syphilis link - continuing to grapple with the problem of how to assess the validity of statistical evidence against traditional pathological and therapeutic definitions.

Locomotor Ataxia, Facial Paresis: Three Diseases and their Relationships, Nov 15, 1887', pp 1 - 25; on p 8.

[137] 'Hence, what use is the concept of syphilis-related ataxia when anti-syphilitic medication is of no use?': Ibid, p 8.

Following Fournier's 1876 work, neurologists were accepting the link with locomotor ataxia far more readily. Fournier had suggested that 80% of tabetic patients had a history of syphilis, and in 1881 the German neurologist Erb corroborated this in a huge numerical study of 1100 tabetics.[140] British neurologists, enthusiastically conducting their own studies, were soon quoting analogous figures; and such evidence was quite rapidly accepted as requiring a reformulation of syphilis.[141] William Gowers acknowledged that tabes was clearly not pathologically a syphilitic disease; neither was it influenced by mercury. However, ' ... such theoretical considerations must yield to facts, and instead of denying that this or that lesion can be produced by a given cause, we may have to widen our view of the operation of that cause.'[142] Admitting the obscure nature of the link, he concluded that tabes was a degenerative sequel of syphilis, exacerbated by factors such as fatigue, injury, or sexual excess.[143]

The relative ease of acceptance of the link highlighted differences between the young neurological and the psychiatric professions. Practically, statistical studies of tabes were far easier to carry out, since patients were gathered in small specialist hospitals such as the National Hospital at Queen Square. Evidence of a syphilitic

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[138] Ibid, p 10.
[139] Corbin, 1995, p 120.
[140] Erb, like Fournier, was evangelical about the value of statistics. In the introduction to his Die Aetiologie der Tabes (1891) he stressed that if an aetiological agent didn't immediately produce symptoms (as with syphilis), then clinical material must be statistically evaluated: 'It is a lack of understanding for the methods of inductive research if some authors want to exclude statistics from investigation...'.Quoted in Peller 1967, pp 36-7.
[141] See, for example, W R Gowers, 'Syphilitic Neuroses', BMJ, Mar 1, 1879 pp 303-5; T Buzzard, Clinical Lectures on Diseases of the Nervous System (Philadelphia: Blakiston, Con & Co, 1882), p 210; Gowers, 1886-8, pp 288-9.
[142] Gowers, 1886-8, p 289.
[143] Ibid, p 315.

history was easier to obtain since patients were usually sane and manageable. It is also possible that tabes fitted more readily into the syphilitic framework because it had far stronger and more traditional associations with venereal excess than did GPI. From the eighteenth century the spinal cord had been popularly linked with sexual function; wasting of the cord was thus linked with 'masculinity misapplied' - sexual incontinence or masturbation.[144] These associations lingered despite the more circumscribed clinical appropriation of 'tabes' during the 1840's.[145] As Romberg noted in his classic description of 1846: 'The majority [of patients] do not complain much ... If they are members of the higher classes, they anxiously endeavour to conceal their loss of motor power, in order to avoid the evil reputation of being affected with tabes dorsalis.'[146] Romberg refuted the idea that loss of semen was of much importance in causing the disease; however he conceded that '...When combined with hyperstimulation of the nerves to which sensual abuses give rise, it not unfrequently favours the origin and encourages the development of the disease after it has commenced.'[147] Although Gowers sometimes played down the significance of sexual excess, he also associated it closely with certain symptoms of the disease such as optic nerve atrophy.[148] Paradoxically, a minority of commentators evidently saw this association as a confounding factor - arguing that sexual excess per se caused tabes, and that syphilis was only incidentally linked to sexual excess.[149]

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[144] Schiller, 1976. This connection was extended to other forms of paralysis such as progressive hemiplegias and paraplegias; and cases reported in the literature routinely noted the presence or absence of sexual excess.
[145] As described in Ch 1, p ? , above.
[146] Romberg, 1853, p 397.
[147] Ibid, p 398.
[148] Gowers, 1886-8, p 31.
[149] See Buzzard, 1882, p 211.

During the 1880's, in contrast, asylum records and written accounts continued to concentrate upon the traditional factors - both admirable and blameworthy - of fast living in general paralytics.[150] There were exceptions to this, as a handful of alienists took notice of continental views of the prime role of syphilis.[151] When this 'darkest Africa of psycho-pathology' was discussed by the Medico-Psychological Society in 1887, however, Thomas Clouston insisted that there was no connection between the two diseases, and met with little enthusiastic opposition.[152] Whilst Margaret Thompson would suggest that this denial was due to a profound horror of the degenerative implications of syphilis in psychiatry, Clouston's claimed reasons were pragmatic: a lack of clear statistical evidence to support what was still regarded as a major reconception of the syphilitic domain.[153]

Alienists seemed to cling tenaciously to multifactorial causal explanations despite their organic aspirations. This, complained some, led them to ignore the evidence which their German and Dutch counterparts noticed. It was commonly pointed out that researchers in other countries found antecedents partly because they were taught to look thoroughly for them; British doctors, it was urged, should be primed in the same way. As a reviewer of the 1895 Commissioners' Report

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[150] See, for example, the 48th Commissioners' Report for the years 1888-92. Whilst alcoholic excess and moral causes - mainly business setbacks and anxiety - took first and second places (25% and 17% respectively), venereal disease was mentioned in only 3.9% of male cases of GPI - a larger percentage than was assigned to other mental diseases, but small nonetheless. In contrast heredity took first place for other forms of insanity: Review of the 48th report of Commissioners in Lunacy, 19th June, 1894; reprinted in J Ment Sci, 41, 1895, p 108.

[151] See, for example, W McDowall's translation and commentary on A Foville, 'On the Relation between Syphilis and General Paralysis. The Difficulty of Distinguishing General Paralysis from certain Syphilitic Changes of the Brain', J Ment Sci, 1880, 5, pp 455 - 467. McDowall placed emphasis on the fact that Quaker patients at the York Retreat had a very low incidence of GPI.

[152] The phrase is Jacobson's: Jacobson, 1892, p 175; 'Notes of the Medico-Psychological Association', J Ment Sci, 1886, 31, pp 610 - 612. See also 'Notes and News of the Medico-Psychological Association of Great Britain and Ireland', J Ment Sci, 1892, 38, pp 318 - 332; p 324.

remarked: '...There is too great a proclivity among medical superintendents blindly to discard the syphilitic theory of the origin of this malady in favour of the whole of the other causes...'[154] The alienist Wilson - who himself painted a multi-faceted picture of the typical sufferer - wryly corroborated this: '... A course of reading on the aetiology of general paralysis would incline one to believe that there is no evil under the sun ... that may not sufficiently account for the onset ...'[155]

The statistical evidence itself did not change; neither did the practical difficulties of carrying it out. By 1901 the figures still showed 'an absurd want of accord', with percentages of syphilitic antecedents, quoted from a variety of European studies, ranging from 1.6 to 94 %.[156] Few of these studies were British: it was common wisdom that 'of course medical officers in asylums have far too much to do, and they cannot give the time to the investigation that is necessary to make accurate statistics.'[157] What did change was the openness to look for a syphilitic history in patients; and the willingness to assume that such a history was frequently present even when not revealed. Rather than clinical evidence, then, it was the reformulation of syphilis which Fournier catalysed - first hereditary syphilis, then parasyphilis - that brought about this receptivity. Where many alienists writing in the early 90's allowed syphilis only a small part to play in GPI, by the turn of the century most claimed that

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[153] Thompson, 1985.
[154] Review of the 48th report of the Commissioners in Lunacy ... , J Ment Sci, 1895, p 108. Jacobson, discussing national differences in receptivity, identified the Scandinavians as espousing 'pure causality'; the Germans as moderates; and the British and French as anti-syphilitic: Jacobson, 1892.
[155] Wilson, 1892, p 30.
[156] Stoddart, 1901, pp 444-5.
[157] F W Mott, 'Relation of Syphilis to Insanity', delivered at the Annual meeting of the MPA, London, 1899, published in J Ment Sci, 1899, 45, pp 683 - 699; on p 684. Small, private asylums generally yielded higher figures of antecedents: this was attributed to doctors having more time for research, and to the greater reliability of evidence from accompanying relatives.

it was probably the prime cause. In 1899 Mickle described the death of traditional ideas of syphilitic provenance: '... I have gradually come to see that we must alter our opinions with regard to what we admit to be syphilitic lesions. Gummatous lesions were the so-called syphilitic lesions ... but over and above those there are other lesions which we must admit to be syphilitic, although they have not that apparent character ... Thus, having widened the field of syphilitic lesions, one can account for everything found - every possible variety of lesion in general paralysis, the varieties of degeneration, or atrophy, and inflammation.'[158]

Once the tide of opinion had changed, additional evidence for the link was increasingly cited.[159] Analogies were noted between the geographical, class, and gender distributions of syphilis and of GPI. The identification of the disease in children was considered important: these patients were assumed not yet to have been exposed to the stresses of civilisation, and high numbers of syphilitic antecedents - whether congenital or acquired - were far more striking than in adults.[160] The German psychiatrist Krafft-Ebing conducted inoculation experiments in 1899 which showed, he claimed, that GPI patients must already be infected with syphilis. Perhaps most important, the accepted syphilis-tabes link, and the apparent clinical relationship between tabes and GPI, provided a strong - if often circular - argument. In his Les Affections Parasyphilitiques, Fournier argued that one of the main similarities between the two diseases was that both were caused by syphilis; on the other hand, to

[158] Quoted in Ibid, p 695; see also W R Dawson, 'The Relation of Acquired Syphilis to Insanity', J Ment Sci, 1898, 44, pp 277 - 290.
[159] For a consideration of all these arguments, see Mott, 'Relation of Syphilis to Insanity', 1899.
[160] Clouston made one of the earliest identifications of juvenile paresis: T S Clouston, 'A case of general paralysis at the age of sixteen years', J Ment Sci, 1877, 23, p 419.

demonstrate the close connection between the two - he claimed - was to indirectly demonstrate the connection between GPI and syphilis.[161] The theory of parasyphilis, he concluded, finally brought the two diseases together: '... as two topographic modalities of a single ... morbid process, as two branches of the same trunk, as two geographical localisations of the same malady ...'[162]

'The Apotheosis of Selfishness': GPI as Degeneration

The neuropathologist Frederick Mott (1853 - 1926) has traditionally been identified as most instrumental in bringing about acceptance of the syphilitic link in Britain.[163] At his death, it was noted: 'His greatest achievement was the definite proof that GPI was due to infection and was part of a general disease, and had nothing to do with nervous inheritance or with the strains and stresses of an over-active life. In this way was removed the disability previously attached to descendants of a general paralytic, and the disease itself entered into the domain of those which are preventable, and possibly, with further research, susceptible of cure...'[164] Although Mott was the first to admit that his aetiological ideas were not original, and had lagged behind those of the continent, he was an influential public figure, and probably indeed swayed medical opinion in Britain. His approach to the problem of the link, however, was very different to that of both earlier German and Scandinavian authors, and of Fournier.

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[161] Fournier, 1894, p 225; pp 236-7.
[162] Ibid, p 231.
[163] See, for example, Oriel, 1994, p 55; E H Pryor, Claybury: A Century of Caring (Suffolk: The Mental Health Care Group, Forest Healthcare Trust, 1993) p 69.

Educated at University College London, Mott studied with experimentalists such as Victor Horsley and E A Schafer, and developed a keen interest in brain neurophysiology.[165] Following posts in physiology, pathology, and clinical medicine at Charing Cross Hospital, he was appointed chief pathologist to the Central Laboratory of the London County Asylums which was established on the Claybury Asylum site in 1895. During the following years he carried out copious research into the effects of structural and functional brain disorders on the mind, publishing much of his work in his own journal, the Archives of Neurology. Mott's first major statement on the relation between GPI and syphilis appeared in 1899.[166] Five years before, he commented, he had thought that GPI was due simply to stress. His opinions had not been changed by the evidence of statistics, since he and his colleagues had little opportunity for numerical studies.[167] Instead his conception derived from the neuropathology of GPI, which he interpreted on the basis of histological and chemical work upon 'degeneration', or 'decay', of the neurones - the subject of his 1900 Croonian lectures.[168] Such neuronal degeneration occurred in GPI, he argued, as a result of systemic poisoning - mainly from syphilis, but also from other forms of civilisation-induced stress: 'The syphilitic toxin is one of many factors in the production of the degenerative process: I do not believe it is essential ... But I

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[164] Prof E H Starling, Address at the Memorial Service for F W Mott, printed in J Ment Sci, July 1926, 72, pp 317 - 320; on p 319.
[165] For a comprehensive account of his work, see A Meyer, 'Frederick Mott, Founder of the Maudsley Laboratories', Brit J Psychiatry, 1973, 122, pp 497 - 516.
[166] Mott, 'Relation of Syphilis to Insanity', 1899; Idem, 'Observations upon the Aetiology and Pathology of General Paralysis', Archives of Neurology, 1899, 1, pp 166 - 203.
[167] Mott, 'Relation of Syphilis to Insanity', 1899, p 691. The exception to this was the convincing evidence of juvenile GPI: F W Mott, 'Notes on 22 Cases of Juvenile General Paralysis, with 16 Postmortems', Archives of Neurology, 1899, 1, pp 250 - 327.

do say ... that syphilis is the most important factor by reason of its prevalence, its persistence and its potency in producing devitalising effects upon structures which are incapable of repair, and incapable of regeneration.'[169] Over the following years, Mott would rephrase the mode of action of syphilis, suggesting, for example, that the virus 'damaged the durability of the neurones, so that systems or communities die prematurely ...'; or that it reduced the vital energy of the nerve cells, making them vulnerable to other insults such as 'brain fag' and alcohol - thus '... systems of the neurons may, under the influence of stress in one form or another, die prematurely...'[170]

Throughout his career Mott took a keen interest in the hereditary causes of mental disease. One of his major projects was to institute an ambitious card system of patients and relatives in the London County Council asylums, through which he gleaned statistical details of disease pedigrees. He concluded that certain forms of insanity - particularly manic-depressive, delusional, and dementia praecox - had a marked tendency to direct inheritance or to inheritance through the neuropathic temperament.[171] He was not, however, a thorough-going degenerationist: he did not believe in the natural decay of nations, and placed great emphasis upon 'anticipation' - the progressively earlier onset of mental disease through generations which led to a regeneration of the stock. His prescription to restore the vigour of the English race

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[168] F W Mott, The Croonian Lectures: The Degeneration of the Neurone (London: Bale and Danielsson, 1900).
[169] Mott, 'Relation of Syphilis to Insanity', 1899, pp 688-9.
[170] F W Mott, 'Morison Lectures: The Pathology of Syphilis of the Nervous System in the Light of Modern Research', Archives of Neurology and Psychiatry, 1909, 4, pp 13 - 57, on p 43; Idem, 'Relation of Syphilis to Insanity', 1899, p 687.

was 'due pruning of rotten branches and due hoeing up of weeds'.[172] His membership of the British Eugenics Society addressed the first of these issues: specific disease eradication through control of breeding - although he was cautious about causing class antagonism through extreme measures such as sterilisation. The second issue referred more vaguely to eradication of the acquired influences of civilisation: alcoholism, selfishness and 'luxury'.

Mott was clear that the neuropathic taint was peculiarly unimportant in GPI itself, although his data seemed to suggest a small hereditary component. This he explained upon the basis of inheritance of a sexually ardent temperament (increasing a man's exposure to syphilis), or inheritance of an 'immune hypersensibility' which caused an over-reaction to syphilis infection.[173] Nevertheless, both his pathological description of the disease and his idea of the action of the syphilis toxin upon a nervous system debilitated by the stresses of civilisation were strongly suggestive of degenerative imagery. In addition, his disease pedigrees freely included general paralytics as indicators of poor ancestral stock. Such imagery epitomised a flexible depiction of GPI which became increasingly prominent over the turn of the nineteenth century. Whilst traditional causal associations - particularly alcoholism - were naturally coloured by degenerative interpretations, it was the link with syphilis, the insidious racial poison, that most encouraged the new perception. In consequence, the redeeming masculinity of the general paralytic was increasingly submerged: GPI

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[171] See F W Mott, Huxley Lecture, 1910: 'The Hereditary Aspects of Nervous and Mental Diseases', The Lancet, Oct 8, 1910, pp 1057 - 1064, and Idem, 'The Neuropathic Inheritance', J Ment Sci, 1913, 59, pp 222 - 263.

became the archetypal danger of the civilised collective, rather than a disease which spoke of how the individual man had lived. This change ran in parallel with alarming over-crowding of asylums; and in many ways the imagery of GPI was drawn more and more from the working-class asylum patient rather than from the private patient. It has been observed that the burden of blame for syphilitic degeneration focused most intensely upon men at the turn of the century: GPI - quintessentially the 'masculine' insanity - provided strong corroboration of this.[174]

George Savage, superintendent of Bethlem Asylum between 1879 and 1889, and one of the more enthusiastic of British degenerationists, came to believe firmly in the GPI-syphilis link several years before Mott. In 1886 he suggested that syphilis might be implicated, but allowed it only a minor role. His discussion of the potential general paralytic patient - whilst paying due attention to the dangers of dissipation - presented a broadly sympathetic image of the susceptible individual which stressed his positive masculinity: 'It appears as if among the strongest, most thoughtful, most energetic and useful of men, this disease has its richest harvest'.[175] Six years later he considered syphilis the most important cause of GPI, and attributed this change in opinion to statistical studies which he had carried out amongst his private patients. The degenerative view-point, however, was central to his understanding of the link - and this revealed itself in a shift of emphasis in his writing. Savage had turned his attention away from the individual and towards the collective dangers of the disease,

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[172] Mott, 1910, p 1064. See G R Searle, 'Eugenics and Class' in C Webster (ed), Biology, Medicine and Society 1840 - 1940 (Cambridge: Cambridge University Press, 1981), pp 217 - 242; also Soloway, 1982.

[173] Mott, 1913, p 257.

focusing upon the problem of the working classes, and the deleterious effects of civilisation within the urban environment: 'In the crowded cities of pushing peoples the disease grows rankly ... With the press of population to towns and cities, the increasing relative fewness of our rural populace, the tension of modern and diffused activity and debauchery, the decrease of leisure and deliberateness, there can be little doubt that general paralysis is rising.'[176] Degenerative images of ruin and decay now permeated many aspects of his discussion of GPI - as when he compared the patient's brain to a neglected house or to a piece of rotting fruit.[177] On an individual level, it was expressed as a more intense focus upon moral degeneration in the early stages of the disease: 'In general paralysis of the simplest form there is, throughout ... solely or chiefly a dementia, a decay, or destruction, of the moral intellect and emotional faculties ... the acts cease to be guided, as formerly, by religion, altruism, sense of morality or duty, patriotism, love of family, of truth, of friendship, of beauty ...'[178] On a pathological level, it was expressed through descriptions of the mode of action of syphilis: '[Syphilis'] tendency is to start a process of degeneration which ultimately produces the ruin we recognise as general paralysis ...'; '... the degeneration follows on the predisposing [local] condition. The fungi grow on the dungheap, but do not form it.'[179]

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[174] Spongberg, 1998, p 255: 'It was generally men who showed symptoms of GPI and in this capacity they became symbols of moral degeneracy...'
[175] Savage, 1886, p 27.
[176] G Savage, 'General Paralysis', in D Hack Tuke (ed), A Dictionary of Psychological Medicine, 2 Vols (London: J&A Churchill, 1892), Vol 1, pp 519-44; on p 535.
[177] G H Savage, 'The Warnings of General Paralysis of the Insane', BMJ, April 5, 1890, pp 777 - 780.
[178] Savage, 1892, Vol 1, p 521.
[179] Ibid, Vol 2, p 1257; Idem, 1890, p 780.

The idea of degeneration - as numerous historians have demonstrated - was nebulous enough to reach far beyond the remit of hereditary theory.[180] Such flexibility allowed GPI to be fitted quite effortlessly into the degenerative framework, despite the fact that it was predominantly associated with acquired syphilis rather than the hereditary disease. This common stock of ideas was often regarded as blatant confusion by contemporaries - as demonstrated by the arguments about two well-publicised literary works of the period. Ibsen's rather confused portrayal of an 'heredo-syphilitique', cursed by the sins of his parents, captured well the popular association of insanity and hereditary vice. The play in question, Ghosts, was published in Norway in 1881, at a time when the GPI-syphilis link was already established in Scandinavia; by 1891, when it received its single scandalous performance in Britain, the possibility of a link would have been familiar to doctors, although probably not widely to the general public (note the narrative of Randolph Churchill).[181] The portrayal of the insane Alvin was certainly contradictory according to medical wisdom: he had reached his adulthood with no signs of sickliness or hereditary syphilis; instead his insanity had been preceded by a period of dissipation which might well have been associated with GPI. His clinical symptoms, however, were not typical of the disease - he suffered, for example, from depression and apprehension rather than paralysis, dementia, or delusions of grandeur. Clearly Ibsen's intentions were literary rather than scientific; but Max Nordau, in his

[180] For a discussion of Lamarkian ideas of degeneration, and Weismann's refutation, see Nye, 1982; Soloway, 1982.

[181] H Ibsen, Four Major Plays, transl J McFarlane (Oxford: Oxford University Press, 1981). For popular reaction to the play, see Spongberg, 1998, pp 245-6. Spongberg suggests that part of the

polemical against degeneracy in the arts, took great delight in ridiculing this anti-realism: 'Ibsen freely invented his invalids ... [inventing] unknown maladies ... which he churns out for poetic effect.'[182] Nordau's work itself did not make a deep impact in Britain outside literary circles; but it too provoked a number of sweeping attacks. GPI played a part in one such debate, in which the critic Talbot ridiculed Nordau's assumption that the French poet Baudelaire must have been a degenerate simply because he died of the disease.[183] Talbot was concerned to strictly limit the definition of degeneration to a hereditary or congenital condition which manifested itself as a developmental disorder. GPI, he objected, was acquired by fully-developed adults: it was not a developmental disorder; was seldom found in degenerates, and 'never in idiots'.[184] Talbot's objections emphasised the very point that syphilis could be cast as a profound and insidious degenerative influence quite apart from its strictly congenital effects; equally that many commentators were still eager to counter claims of degeneracy in GPI with a defence of its victims' calibre.

Nevertheless, Savage's writing was representative of a considerable strain of thought over the turn of the century, as the pathological, clinical, and moral aspects of GPI were conflated. In such writings the pejorative associations of syphilis now applied in full force to the general paralytic. His sexual excess had never been over-emphasised in the British literature; and when mentioned it had often been

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hostility related to its portrayal of 'the male syphilitic [rather than the female] as model of pathological sexuality', p 246.

[182] M Nordau, Degeneration, transl from 2e (Lincoln and London: Univeristy of Nebraska Press, 1968), p 285. German discussions of Nietzsche's illness - which I do not enter into here - encapsulated the conflation of disease, character, and degeneration which general paralysis suggested. For an introductory account of the possible effect of GPI upon Nietzsche's writings see R Hayman, Nietzsche: A Critical Life (London: Weidenfeld and Nicolson, 1980), esp pp 319-50.

[183] Nordau, 1968, p 354.

euphemised as a 'gay life'. Increasingly, though, sexual excess became the symbol which linked syphilis, degeneration, and GPI. Just as Mott's careful neuropathological work described degeneration - in both its syphilitic and its cerebral aspects - on an organic level, so shifts in documented symptoms suggested it on a clinical level: alienists frequently claimed that the disease was less florid than in former years, and was increasingly marked by slowly worsening dementia, rather than by glorious delusions of wealth and generosity. Interestingly such a trend, according to contemporary ideas, could be viewed as the development of a weaker, more 'womanly' form of the disease. Indeed female general paralytics became less invisible from the turn of the century: the increasingly accepted link with syphilis naturally suggested a focus upon prostitution; in addition, GPI appeared to be rising precipitously amongst women from the 1890's. These trends, combined with the traditionally lower-class associations of the disease in women, meant that GPI could be entered into a degenerative framework even more readily than was the case for men. A twentieth-century report clearly reflected ideas about the comparative merits of the male and female characters. Male general paralytics, it suggested, were recruited from all social grades, and were frequently productive members of society; women, in contrast, tended to come from 'the unfortunate class of women, many of whom are mental defectives ...' Correspondingly the male general paralytic usually possessed 'a well-convoluted brain of above the average; whereas the female

[184] E S Talbot, Degeneracy: Its Causes, Signs, and Results (London: Walter Scott, Ltd, 1898), p 326.

paralytic possessed very frequently a small brain of simple convolutional pattern, indicative of an imbecile ...'[185]

GPI was in fact perceived to be rising out of proportion to the rest of insanity amongst both sexes at the turn of the century, and this intensified the apocalyptic images which were increasingly applied to it.[186] In 1896 the Scottish psychiatrist Stewart published one of the most negative accounts of the disease yet written. The rise of GPI, he suggested, represented 'a reversion to a lower and more hopeless form of brain disease, a diminishing vitality, a lessening power of resistance, and an increasing tendency to premature and rapid racial decay.'[187] Although he noted that hereditary influences were less prominent in GPI than in other insanities, his broader view of degeneration enabled him to blame drunkenness, sexual excess and moral decadence for a reduction in racial resistance of which GPI was a prime manifestation. To Stewart, the individual history of the illness provided a satisfying metaphor for the deterioration of society: 'The opening chapter is moral decadence; the closing acutely rapid physical and intellectual degeneration and premature extinction.'[188] In this bleak interpretation, the previously positive associations of the paralytic patient were completely lost: 'Selfish indulgence, lustful gratification, insatiable animalism, general sensuality and fastness, these are to a very large extent the 'grand-parent manufactory' of the evil ... General paralysis ... is the apotheosis of

[185] Asylum reports, J Ment Sci, 1915, 61, pp 300-1.
[186] I return to both of these themes - the rise in GPI and the change in clinical forms - in the context of historical epidemiology, in Chapter 5 below.
[187] Stewart 1896, p 774.
[188] Ibid, p 776.

selfishness.'[189] Similar sentiments surfaced in the discussion of Stewart's paper: 'The President [Julius Mickle] said ... 'General paralysis was particularly liable to occur in cities ... with a large aggregation of persons who were in some cases the sweepings of creation and lacking self control'.[190] The asylum commissioners' report from the same year was as forthright about the causes of the disease: 'The origins [of GPI] lie in self- indulgent yielding to and perversion of natural instinct, and excesses of all kinds...'[191] At the dawn of the twentieth century, the Claybury psychiatrist Robert Jones' address to the British Medical Association, on the subject of insanity and civilisation, articulated the fear that GPI epitomised the crisis of 'mental wreckage and physical degeneration' resulting from rising syphilis, alcoholism, idleness, poverty, and crime.[192] Such suggestions that the worst, rather than the best, of men succumbed to the disease represented a striking change from dominant descriptions of only a few years previously: what had once been regarded as manly ambition, ego, or 'fastness' was now seen as pernicious and selfish degradation on an alarming scale.

Into the Twentieth Century: 'Syphilisation and Civilisation'

At the beginning of the twentieth century, then, it seemed to British doctors that the evidence surrounding GPI was sufficient to 'convict syphilis in any court of law.'[193] Once the idea had taken hold during the 1890's there was little resistance to the

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[189] Ibid, p 776.
[190] From 'Notes and News: A Discussion of Dr Stewart's paper on General Paralysis', J Ment Sci, 1896, 42, pp 882-4; on p 882.
[191] 'Asylum reports from Glamorgan', J Ment Sci, 1896, 42, pp 203-4.

doctrine - and certainly no evidence of the stormy boundary disputes that seem to have occurred in France. This is not surprising considering the contrasting states of specialisation between the two countries. In France Fournier was leading a charismatic campaign to create venereology as a credible discipline. Britain lacked such a vocal champion: Jonathan Hutchinson, the major syphilis spokesman during the second half of the century, was a general surgeon who remained steadily anti-alarmist about the ravages of the disease. He also remained suspicious of specialisation - although following the pattern of the period he held part-time appointments at a number of specialist hospitals.[194] Where Fournier advertised the widening domain of his chosen disease, Hutchinson played it down: 'It would be absurd to speak of syphilis as in the main a bugbear, but the impressions derived from my own experience as to its curability and remote results incline me to suspect that the gross exaggerations prevalent respecting it cause more misery than is produced by the disease itself.'[195]

Such a strain of caution about the ravages of syphilis may well have played a part in early resistance to accepting the syphilis-GPI link: as the physician Coutts complained in his 1898 discussion of third-generation transmission: 'The ills of syphilis are numerous and far-reaching enough without any extra fanciful additions [parasyphilis] being credited to them ... In writing of a disease like syphilis in which it

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[192] R Jones, 'The Development of Insanity in Regard to Civilisation', J Ment Sci, 1903, 49, pp 776-7.
[193] Stoddart, 1901, p 454.
[194] Hutchinson, 1946, esp p 84. For general accounts of specialisation, see R Stevens, Medical Practice in Modern England: The Impact of Specialization and State Medicine (New Haven and London University Press, 1966); M J Peterson, The Medical Profession in Mid-Victorian London (Berkeley and Los Angeles, Ca: University of California Press, 1978).
[195] J Hutchinson, Syphilis, enlarged 2e (London: Cassell and Co, 1909), p xii.

is said 'everything is possible' it is well not to be too dogmatic...'[196] Astley Cooper and Marshall, also general surgeons who held appointments at the London Lock hospital, were similarly unterritorial about their field: although both routinely called for State intervention to control syphilis over the turn of the century, neither overtly linked these pleas to specialty expansion.[197] Not until the 1910's, when a wave of propaganda led to the establishment of Government venereal disease clinics, did doctors start to think in terms of a legitimate venereology discipline.[198] The British Journal of Venereal Diseases, established in 1925 (three years after the corresponding medical society) was committed to scientific credibility; nevertheless, it was still ambivalent about the practicality of specialisation in a disease which manifested itself over such wide areas of medicine.[199] In this climate, knowledge and opinions flowed freely between psychiatrists and physicians or surgeons interested in syphilis.

It is also true to say that, whilst the syphilis link lent powerful new associations to GPI at the turn of the century, it did not represent a wholesale revolution in aetiological ideas. Rather it was incorporated into the traditional nineteenth-century framework, and the new model was fluid enough to encompass a range of viewpoints about the centrality of its rôle. Krafft-Ebing's often-quoted phrase: 'Syphilisation and civilisation' encapsulated this: syphilis had become the

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[196] J A Coutts, 'On a Case of Supposed Transmission of Syphilis to the Third Generation', The Lancet, Jan 22, 1898, pp 217-8.

[197] See, for example, A Cooper, Syphilis (London: J&A Churchill, 1895), p 430 onwards.

[198] As in France, dermatology fared far better as a specialty during the nineteenth century and acted as an umbrella for venereal diseases: journals of skin diseases were appearing, intermittently, by the 1860's; and teaching courses and the first chair of dermatology had appeared by the 1870's (championed by Erasmus Wilson). Not until 1917 did a journal appear with 'syphilology' in the title: the British Journal of Dermatology and Syphilology - organ of the British Association of Dermatology.

focus of attention, but alienists still referred freely to alcoholism, mental stress, ambition, sexual excess, and - sometimes - neuropathic heredity when discussing GPI. Even injury to the head - a relatively frequent nineteenth-century factor - was still sometimes quoted; and after the introduction of the Workmen's Compensation Act, patients and relatives themselves had an incentive to claim this as the prime cause.[200]

This retention of multifactorialism confirms the suggestion that the impact of germ theory upon psychiatric thinking at the end of the century was hardly great; and it would continue well after laboratory medicine had apparently provided a definitive answer to the question of syphilitic aetiology.[201] Mott's conception, as I have noted, relied upon the action of syphilis upon a nervous system weakened by the stresses of civilisation: 'The struggle for existence is falling more and more upon the nervous system, and thus it becomes the locus minoris resistientiae ...'[202] The approach was clearly well-suited to the degenerative interpretation of the disease. It was also suggested, on a pathological level, by Fournier's concept of 'parasyphilis', which did not elucidate exactly how syphilis might act. Thus alienists still spoke of syphilis preparing the cerebral soil for further injury - models which required further factors to complete the damage. Others spoke of accessory factors 'giving direction to' the action of syphilis: hence the suggestion that brain-workers were prone to GPI, whilst

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[199] See H D Rolleston, Introduction to Brit J Ven Dis, 1925, 1:1, pp 1 - 10; J H Sequeira, 'The Place of Venereal Diseases in the Medical Curriculum', and discussion, Brit J Ven Dis, 1927, 3: 3, pp 196 - 218.

[200] See Mott's comments in 'The Differential Diagnosis of Syphilis and Parasyphilis of the Nervous System', Lancet, Nov 18, 1911, pp 1392 - 1397; on p 1396.

[201] See Bynum in Berrios and Freeman, 1991, p 172.

[202] F W Mott, 'The Relation of Syphilis to Public Health, including Congential Syphilis', The Lancet, June 15, 1912, pp 1611 - 1614; on p 1611.

physical labourers were prone to tabes. Such flexibility in many ways softened the radicalism of the syphilitic aetiology.

Doctors who did voice objection to the theory usually referred to two clinical problems: that only a small percentage of syphilitics developed GPI; and that GPI was rare in certain uncivilised countries which nevertheless had a high incidence of syphilis. But multifactorialism, accepted even by those enthusiastic about the link, was sufficient to explain away these arguments. Traditional factors, it was reasoned, must provide the missing factor which induced some syphilitics to become general paralytics. The second problem was explained perfectly by the image of the uncivilised savage - peculiarly free from the curse of Western insanity. As the British medical officer for an asylum in Singapore remarked in 1900: 'It has seemed to me that our natives cannot be expected to be the subjects of general paralysis with their simple life, few or no worries, and the fact that there is no struggle for existence among them. In a country where there is no cold, but little indulgence ... in alcoholic excess, where food and lodgings are cheap, and the least possible clothing required, a disease originating in anxiety, mental worries, and great excesses, is little likely to develop, notwithstanding the fact that the ... preliminary disease be present.'[203] In a similar vein Mott explained the corresponding paradox in the Arab countries: 'Syphilis is extremely prevalent; but when the Arab gets it he simply says 'Kismet',

[203] W Gilmore Ellis, 'Syphilis and GPI', J Ment Sci, 1900, 46, pp 208 - 9; on p 209.

and does not worry about it. He is quite satisfied, lives a very frugal life, and there is not the same stress as in this country ...'[204]

[204] Mott, 'Relation of Syphilis to Insanity', 1899, p 690.

CHAPTER 3

'GPI' BECOMES 'NEUROSYPHILIS'

Introduction

In 1914 a bewildered doctor noted: 'The discovery of the spirochaete and of the Wassermann [test] ... has thrown the whole subject of syphilis into the melting-pot, from which new conceptions of the disease are emerging ... These are still malleable, and fresh investigations are daily entailing their remodelling...'[1]

This comment reflected profound changes in the knowledge of, and response towards, syphilis between the turn of the nineteenth century and the second decade of the twentieth. At the beginning of this period the disease was firmly established in the public eye as a dangerous social scourge whose boundaries seemed to be increasing. The addition of hereditary disease, GPI, and tabes to its list of associations confirmed the impression that its clinical manifestations were insidious, protean, and potentially lethal. In France, Fournier stimulated a relatively candid and energetic response to this threat through his 'Société de Prophylaxie Sanitaire et Morale', established in 1901.[2] British initiatives were slower in coming. The Edwardian period was marked by the implicit inclusion of syphilis in debates about

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[1] H Armstrong, 'On Some Clinical Manifestations of Congenital Syphilis', BMJ, May 2, 1914, pp 958 - 961; on p 960.

[2] As in other European countries, this national organisation was stimulated by the First International Conference for the Prophylaxis of Syphilis and of Venereal Diseases, held in Brussels in 1899. For a discussion of the Société's aims, see A Corbin, 'Le péril vénérien au début du siècle: prophylaxie sanitaire et prophylaxie morale', Récherches, 29 Dec, 1977.

degeneration and racial efficiency, but an apparent reluctance to implement public initiatives to combat the disease.

Yet syphilis was rapidly entering the embrace of laboratory medicine, a development which offered the opportunity to nudge it from the sphere of the moral to the sphere of the scientific. Since the dawn of Pasteurian medicine, doctors had tried to identify the responsible organism, but - as seemed fitting for this notoriously elusive disease - had continually failed. Finally in 1905, Schaudinn (a microbiologist) and Hoffmann (a venereologist) announced from Berlin that they had observed a pale, fragile spirochaete associated with syphilitic lesions.[3] 'Treponema pallidum' was rapidly accepted as the primary cause of syphilis - although its resistance to laboratory culturing meant that it never fulfilled Koch's postulates. In the following years, copious experimental studies of syphilis were published, and work upon an immunological diagnostic test for the disease intensified. During 1906 the Berlin Public Health Authority commissioned the Bavarian immunologist Wassermann, together with a number of co-workers, to investigate such a possibility; and a year later the team triumphantly published details of a successful complement-fixation blood test, involving a complex series of interactions between animal and foetal products and human serum.[4] The 'Wassermann test' rapidly entered the day-to-day practice of syphilology - bringing with it both benefits and tortuous theoretical and practical problems.

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[3] F Shaudinn and E Hoffman, 'Preliminary report on the presence of Spirochaetes in syphilitic lesions and in papillomas', English translation in Selected Essays on Syphilis and Smallpox (London: New Sydenham Society, 1906), pp 3 - 15.

[4] A Wassermann, A Neisser, C Bruck, 'Eine Serodiagnostiche Reaktion bei Syphilis', Deutsche Medizinische Wochenschrift, 1906, 32, pp 745 - 746. See Oriel, 1994, pp 77 - 80.

Much has been written about both the socio-political aspects of syphilis, and the general impact of laboratory medicine during the early twentieth century. Most interesting for the purposes of this thesis is the point at which these two themes meet. It is striking that the Wassermann test is practically invisible in accounts of increasing interest and state involvement in venereal diseases during and after the first World War; yet it both stimulated this interest, and was absolutely central to the aims of diagnosis, treatment and surveillance established during the period.[5] Indeed one can argue that the twentieth-century discipline of venereology was created around the Wassermann test. Traditional accounts of laboratory medicine have concentrated upon the theme of triumphant progress; and certainly the test offered ample evidence of this to contemporary doctors. Following its introduction into Britain in 1908 it entered general medical practice almost over-night, and was quickly a central part of syphilis management. Here, we might suppose, was an indication that the majority of doctors were indeed eager to apply a laboratory tool to their clinical practice.

But more recent interpretations of the laboratory revolution lead us to examine the reception of the test more closely. Starting from the laboratory itself, a number of studies have analysed the practices and interests of bench workers and the knowledge claims that they made. Much of this work has been informed by the sociology of knowledge, and has challenged contemporary scientists' insistence that the laboratory allowed nature to 'speak for herself', and thus was - pace the clinic - supremely objective.[6] Moving beyond the location of the laboratory, a major theme has been the

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[5] See, for example, Adler, 1987; Evans, 1992.

[6] See A Cunningham and P Williams (eds), The Laboratory Revolution in Medicine (Cambridge: Cambridge University Press, 1992); J V Pickstone (ed), Medical Innovations in Historical Perspective

nature of the benefits which clinicians received from laboratory tools. Shortt, for example, suggests that laboratory science was exploited by doctors less for its pragmatic value than for it rhetorical value in legitimising the status of the profession: 'Science's catalytic status-conferring power for medicine came more from its use as a cultural than as a technical resource'.[7] More fine-grained accounts have drawn out the interests of different professional groups, highlighting the tensions that practitioners faced in reconciling opposing models of the clinic and of the laboratory. Warner, for example, concentrates upon such tensions in the period of post-Civil War America, and finishes his account at the 1890's, when he suggests that the laboratory model won the battle.[8] An earlier account by Maulitz, however, describes US physicians until World War I still gripped by tensions about the proper boundaries of medical science, and about how far medical decisions should be transferred from the bedside to the laboratory.[9] Several authors highlight the parallel tensions that British doctors faced. Lawrence describes the opposition of London physicians - still wedded to the interests of the 'gentlemanly' profession - to surrendering their clinical prerogative to technological medicine, which smacked of the artisan.[10] This account is based upon rhetoric which, as Jacyna emphasises, can only tell us so much about the relations of the laboratory to the clinic. Jacyna instead bases his study of surgical

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(London: Macmillan Academic and Professional Ltd, 1992); C Lawrence, review of Pickstone 1992 in Med Hist, 1993, 37, 4, pp 449 - 452.

[7] Shortt, 1983.

[8] J H Warner, 'The Fall and Rise of Professional Mystery', in Cunningham and Williams, 1992, pp 110 - 141.

[9] R C Maulitz, '"Physician versus Bacteriologist": The Ideology of Science in Clinical Medicine', in M J Vogel and C E Rosenberg (eds), The Therapeutic Revolution (Philadelphia: University of Pennsylvania Press, 1979), pp 91 - 108.

[10] C Lawrence, 'Incommunicable Knowledge: Science, Technology and the Clinical Art in Britain 1850 - 1914', J Contemporary History, 1985, 20, pp 503 - 520.

practice in Glasgow upon clinical and pathological records, and focuses upon one area of management - breast disease. He concludes that, although many Glasgow surgeons sympathised with laboratory ideals, a stubborn faith in clinical judgement prevented them from integrating pathological authority into their practice.[11] Jacyna hints that during the 1920's Glasgow surgeons were as resistant as ever to the claims of pathology in their practice; and as late as 1937 he identifies overt expressions of antagonism towards the diagnostic role of the laboratory. Clearly, the story of tension between the two models can be extended far beyond the 1910's.

Such descriptions of conflict - both in expression and in practice - are interesting and complicated because they are placed against a backdrop of increasing excitement amongst many about the advances and powers promised by the laboratory. The Wassermann test provides an excellent means of studying these rather elusive aspects of the laboratory-clinic relationship. It elicited a wide range of reactions - and not necessarily in ways that polarised groups of practitioners. Some claimed enthusiastic faith in its powers; some attacked it virulently; but many expressed the need to cautiously balance its evidence with that of the clinic. Debates often centred upon the practice of laboratory workers who developed and carried out the test: problems of the production and negotiation of knowledge - fertile ground for recent historians - were clearly appreciated and fiercely discussed by early twentieth-century doctors.

The Wassermann test in general medicine is the backdrop to the main theme of this chapter: its application to GPI. During the first five years of the century a

[11] L S Jacyna, 'The Laboratory and the Clinic: The Impact of Pathology on Surgical Diagnosis in the

number of laboratory techniques had been applied to the cerebro-spinal fluid of mentally disturbed patients in an effort to refine diagnosis: precipitin and coagulin tests to measure globulin content; differential cell counts to measure plasma cells and lymphocytes. Later, in 1912, the colloid reaction was accepted as a technique which gave a very specific reaction for GPI - the 'paretic zone'. In the same year, two experimentalists identified spirochaetes in the brains of general paralytics - a dramatic confirmation that GPI could be reformulated as 'neurosyphilis'. The Wassermann reaction, however, became the central part of the new scientific investigation into mentally ill patients, and as a practical tool which entered the world of everyday psychiatrists, it was their most potent symbol of the laboratory era. Psychiatrists incorporated the test quite readily into their practice as they used it, first, to clinch the link with syphilis and, second, as a means of diagnosing GPI. Here was a further opportunity - perhaps the most promising yet - for them to bring their specialty closer to the ideals of scientific medicine. 'Parasyphilis' was replaced by the more confident term 'neurosyphilis' - the first convincing aetiological conception of a mental disorder; and some claimed that the boundaries of GPI had been broadened as laboratory diagnosis added to - and often over-ruled - clinical diagnosis.

Most historical accounts of GPI offer us only a traditional interpretation of how the laboratory made its impact - assuming that its tools led to unequivocal advances in understanding and managing the disease, and that it set a successful scientific example which psychiatry could follow.[12] Certainly, psychiatrists made good rhetorical use of the laboratory model. Indeed, there is less evidence of

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Glasgow Western Infirmary, 1875 - 1910', Bull Hist Med, 1988, 62, pp 384 - 406.

antagonism to the use of the test than was apparent in general medicine - although there were objections, particularly after the War, which related closely to the very means of defining GPI. But it is important to balance practitioners' words - which often seem to hang in a vacuum - with an assessment of what was happening in day-to-day practice. In this chapter, therefore, I shall try to assess how the Wassermann test made a practical impact upon the specialty; and will suggest that in many ways the rhetorical importance attached to it outweighed the functional benefits that psychiatrists felt they had gained.

'A Thoroughly Wassermannized Profession': The Test Enters Medicine

In Britain the Wassermann test attracted intense professional interest, and inspired an avalanche of articles in the medical press. Early reports suggested that it became positive in the blood three to six weeks after a person was infected with syphilis, and thereafter became increasingly sensitive until, by the time the secondary stage was reached, roughly 85% of patients yielded a positive reaction. As the disease moved into the latent or tertiary stages, this sensitivity fell to between 50% and 70% of cases. Clearly a negative result could not rule out syphilis; but the most burning question concerned the objectivity of a positive result. If syphilis was the master of disguise and concealment, could the test represent the great revealer? Many early commentators had no doubt about this: 'We are entirely satisfied as to the great value of the Wassermann reaction from the diagnostic point of view, such clinically doubtful cases that give a positive reaction always showing later ... that they were

[12] See, for example, Zilboorg and Henry, 1941.

syphilitic'[13] A 1912 Lancet leader caught the mood of enthusiasm: 'Never more than in the last few years has the medical profession appreciated the importance of this disease to those who have been infected with the virus, to the nation at large, and in fact to the whole human race ... A positive Wassermann is conclusive evidence of the presence of the disease even in the total absence of all manifestations ...'[14] Many doctors, then, quickly accepted that a positive reaction was practically pathognomic of syphilis - and the test's revolutionary function in uncovering obscure cases remained a powerful theme over the following years. Syphilis was a dishonest disease, and syphilitic patients were themselves frequently dishonest: the test's potential as a lie-detector was agreed to be immense.

This ability to reveal appeared most striking in its application to large groups of people, since the test's apparent objectivity rendered it the ideal tool with which to sample and compare different groups of patients. Before and during World War I numerous mass serological studies were performed upon diverse groups of individuals: out-patient attendees; general hospital patients; navy servicemen; prostitutes; pregnant women; juvenile delinquents; families of syphilitics; and those with mental deficiency.[15] Further numerous studies were directed at patients with

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[13] C Gibbs and H W Bayly, 'The Comparative Value of the Various Methods of Anti-Syphilitic Treatment: As Estimated by the Wassermann Reaction and Clinical Observation', Lancet, May 7, 1910, pp 1256-7.

[14] Anon leader, 'Our Knowledge of Syphilis', Lancet, July 20, 1912, pp 163-4; my italics. See also 'Report of the West London Medico-Chirurgical Society: The Laboratory Diagnosis of Syphilis', Lancet, Dec 11, 1909, p 1744.

[15] See for example E Bellingham Smith and AWG Woodforde, 'Communication on Syphilis and Mental Deficiency at the Royal Society of Medicine Meeting, Apr 25, 1911', BMJ, May 6, 1911, p 1054; K Fraser and H F Watson, 'The role of syphilis in mental deficiency and epilepsy: A review of 205 cases', J Ment Sci, 1913, 59, pp 640 - 651; W M Elliott, 'The Wassermann Reaction in Children of the Poorer Classes', BMJ, Feb 21, 1914, pp 431-8; C H Browning, 'Investigations on Syphilis as Affecting the Health of the Community: A Summary of the Collective Examination of over 3000 Cases by Clinical and Serological Methods', BMJ, Jan 10, 1914, pp 77 - 81. For a comprehensive reference

particular pathologies - including liver cirrhosis, arthritis, congenital deafness, and ophthalmic disease - in an effort to establish links with the venereal taint.[16] Hereditary syphilis - one of the most insidious forms of the disease - predictably attracted great attention: 'How far a family may be saturated with syphilis, what the [hidden] effects may be, and through how many generations the poison may still be operative are all questions of great interest ... In the solution of these problems serological diagnosis promises to play a great and, indeed, indispensable part.'[17] The results of these studies were certainly not always dramatic, but many confirmed in bleak statistics that hidden syphilis was at the root of numerous medical and social evils.

The test gave great weight to the fear that the disease was moving from the domain of the visible to that of the invisible; that it was becoming less florid and less lethal, but more hidden and insidious in its manifestations. This in turn stimulated renewed calls for prophylactic measures: '... The test shows us that syphilis has more manifestations than were thought, that we must treat it more thoroughly, suspect it more ... renew our fight against this most direful scourge of modern times..'[18] The St Mary's physician Sir Malcolm Morris was one of the most outspoken protagonists for a Royal Commission on Venereal Diseases during the early 1910's. His concerns, set out in several letters to the medical and national press, were clear: the effect of syphilis upon national vigour; the apparent rise in its nervous manifestations; and the

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to such studies see C H Browning and I McKenzie, Recent Methods in the Diagnosis and Treatment of Syphilis, 2e (London: Constable and Co Ltd, 1924).

[16] Leader: 'The Syphilitic Factor in some Obscure diseases', BMJ, Jan 10, 1914, pp 103-4.

[17] Leader, 'The Wassermann Method and Psychiatry', BMJ, Sept 30, 1911, pp 760-2; p 761.

[18] 'Our Knowledge of Syphilis', 1912, pp 163- 4.

new opportunities offered by laboratory medicine.[19] Three years later, Wassermann studies formed the evidence at the heart of the 1916 Royal Commissioners' report, whose recommendations resulted in the provision of laboratory-equipped venereal disease clinics, 'non-nominative' notification, improvement in medical teaching, and encouragement of health education through bodies such as the National Council for Combating Venereal Diseases.[20]

The most obvious practical use of the test for individual doctors was in initiating and monitoring treatment. The 'arsenicals' salvarsan and neosalvarsan became the mainstays of therapy for syphilis after their introduction from 1909; and although later accounts would be muted in their assessment of the benefits of these drugs, it seemed during the 1910's that they provided great hopes for controlling the disease. Most venereology experts recommended that if an asymptomatic or dubious patient tested positive, anti-syphilitic treatment should be started immediately and vigorously. Yet it was quickly realised that many patients continued to test positive well beyond the point at which standard treatment was stopped. This led to a certain therapeutic pessimism: 'Our conception of the curability of syphilis', wrote the Edinburgh psychiatrist George Robertson in 1913, 'has entirely changed since the Wassermann reaction has been employed to control its treatment. In the past many were unfortunately content to remove the external manifestations and call this a cure ... We know now that while the manifestations of tertiary syphilis respond

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[19] M Morris, 'A Plea for the Appointment of a Royal Commission on Venereal Disease', Lancet, June 28, 1913, pp 1817-19; See also Anon leader: 'Syphilis and the Responsibility of the State', Lancet, June 28, 1913, pp 1810-11.

[20] Royal Commission on Venereal Diseases: Final Report of the Commissioners (London: HMSO, 1916).

wonderfully to salvarsan and mercury, it is not possible in some cases to remove the positive reaction from the blood ... Cure ... is tested, not by the disappearance of all visible manifestations of the disease, but by a permanently negative Wassermann reaction, for anything else is futile.'[21]

On the other hand, the finding galvanised doctors into attempting far more extensive surveillance of patients. The Wassermann test became central to a new definition of 'cure': no longer simply the disappearance of clinical symptoms, it now required a convincing period of negative testing. Early recommendations appeared from the Rochester Row army hospital, where the traditional length of treatment had been two years. The institution of testing for all patients in 1908 revealed that this was inadequate; and a new protocol advocated continuation of treatment for a year after the test had become negative, with checks at three, six, and twelve month intervals.[22] This suggestion was conservative by the standards of later guidelines, many of which insisted that patients should demonstrate up to three years-worth of negative results. Overall the recommendations pointed to a marked increase in the length of treatment and observation for syphilitics, in line with the apparent extension of its territory.[23] Similarly, although many doctors agreed that ideally syphilitics should never procreate, in practice the test led to increasingly more cautious advice concerning

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[21] G M Robertson, 'The Morisonian Lectures, 1913: General Paralysis of the Insane', J Ment Sci, 1913, 59, pp 185 - 221; on p 210, p 213.

[22] L W Harrison, 'Report of the Section of Pathology and Bacteriology on Complement-Fixation in Diagnosis', BMJ, Nov 5, 1910, p 1427. Recommendations for protocols varied slightly: see, for example, H W Bayly, 'The Practical Value of the Wassermann Reaction', BMJ, Nov 5, 1910, pp 1427 - 1438; J E R McDonagh, 'Serum Diagnosis of Syphilis', BMJ, Sept 24, 1910, pp 846-7; Leader: 'Recent Developments in the Diagnosis and Treatment of Syphilis', BMJ, Sept 23, 1911, pp 692-3.

[23] Williams notes that the average in-patient stay for the female London Lock hospital rose from 66 days in 1908 to 121 days in 1912, following the introduction of the test: D I Williams, The London Lock (London: RSM Press, 1997), p 116.

proof of cure before marriage. The combination of promising new drug therapies and a revolutionary diagnostic tool appeared irresistible, and in many ways the development of both was intertwined.[24] New terms entered the medical vocabulary which bore witness to the shift in emphasis: the 'Wassermann-fast patient' who - whilst showing no signs of illness - continued to have positive reactions despite vigorous treatment; 'Wassermann relapses', in which the test reverted to positive after a long line of negative results.[25] A Boots advertisement for a brand of arsenical in 1925 took the new stamp of laboratory authority for granted: ' ... Approved by the Ministry of Health. Stabilarsan has a more permanent effect upon the Wassermann reaction ... than any other arsenical'.[26]

Recommendations in the literature did not, of course, necessarily reflect practice, and doctors commonly complained that the test was not being fully exploited. The 1916 Royal Commission report pointed out that very few syphilitic patients were admitted to London hospitals where laboratory facilities were available, and that out-patient treatment was hopelessly disorganised. Only a third of provincial hospitals, it estimated, had facilities for performing the test; and the alternative - ordering it from small private clinics - was expensive, so that doctors were reluctant to offer it to their poorer patients. Panel patients under the 1911 Insurance Act were not covered for such laboratory procedures - although by the beginning of the War the Public Health Committees of several boroughs were providing free services. The

[24] A study from Rochester Row Hospital, for example, used the test as an integral part of assessing the effectiveness of salvarsan: T W Gibbard and L W Harrison, 'A Summary of Results Obtained by the Use of Salvarsan in Syphilis', BMJ, Sept 23, 1911, pp 679 - 686.

army, it seemed, was one of the few sites where good facilities and strict control over men meant that the use of the Wassermann approached its full potential. Many hoped, however, that the new VD clinics of the post-war period would provide the ideal setting for laboratory surveillance. In 1919 the Ministry of Health issued protocols to the medical officers of each clinic, outlining the standards of cure to be attained. Patients could be recommended for discharge, the Ministry suggested, after: ' ... two years freedom from all signs of disease, either clinical or serological, after suspension of all medical treatment ... a Wassermann test being taken not less frequently than every three months...'.[27] Over the following years, such ideals seemed impossible to attain. As one reader later commented, the protocol would necessitate nine expensive blood-tests and more than sixteen visits to the treatment centre for each patient - but in practice, he concluded, the cost was not prohibitive since patients were generally not attending their follow-up appointments, and few clinics were living up to standards of cure.

Despite the gap between recommendations and practice, the test was clearly imported rapidly into general medicine and had profound effects upon the ways that doctors managed syphilis. A 1914 BMJ leader painted a rosy picture of the fruitful relationship between laboratory and medicine which it represented: 'Here the laboratory worker can join hands with the practical clinician, for the triumphs of the former within the last few years in the elucidation of some of the problems of syphilis

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[25] See Leader: 'The early diagnosis and treatment of syphilis', BMJ, Nov 22, 1913, pp 1395-6; on p 1396: 'Cases are occasionally met with in which the blood reaction returns obstinately to the positive in spite of several intensive treatments. These are the despair of the syphilologist ...'.
[26] Advertisement in Brit J Ven Dis, Jan 1925, vol 1, no 1, front inside cover.
[27] Correspondence from Ministry of Health file, Nov 25, 1919. PRO, Kew: MH 55/180.

have been striking, and the welcome extended to them by the latter hearty ...'[28] But this rather over-stated the case: although the test was received by many as a triumphant tool of science, ambivalence towards the claims of the laboratory was as clear in this field as in any area of medicine considered by historians.

The immunologist Ludwig Fleck analysed the development of the Wassermann test in his stimulating (if esoteric) account of syphilis, published in 1935.[29] Fleck saw its creation as a response to increasing demands for a tool which could define syphilis according to the traditional 'thought style' of bad blood.[30] The resulting concentration of energy and resources was intensified by national competition between France and Germany - a competition which, of course, Germany soundly won. Fleck was fascinated by the tortuous and serendipitous path of this scientific discovery. He described subtle adjustments in serum dosage as scientists tried to achieve an acceptable balance of sensitivity and specificity; and identified a turning point after which the reaction was finally agreed to be useful.[31] Even after a usable test had appeared, it emerged that scientific assumptions about how it worked were incorrect. As Fleck romantically observed: 'From 1907 there were numerous demonstrations that normal organs could produce the reaction as effectively as

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[28] Leader: 'The Syphilitic Factor in some Obscure Diseases', BMJ, Jan 10, 1914, pp 103-4.
[29] Fleck, 1935; see also B G Rosenkrantz, 'Review of L Fleck: Genesis and Development of a Scientific Fact', Isis, 1981, 72: 1: 261, pp 96 - 99.
[30] 'Had it not been for the insistent clamour of public opinion for a blood test, the experiments of Wassermann would never have enjoyed the social response that was absolutely essential to the development of the reaction ...': Ibid, p 77. Fleck's 'thought style' referred to 'the entirety of intellectual ... readiness for one particular way of seeing and acting and no other ...': Ibid, p 64.
[31] Homer Wright's comments about a later modification of the Wassermann demonstrate well the apparent arbitrariness of such decisions: 'This precipitation test is too sensitive. If I tested the blood of patients by this method, I would demonstrate that half of the population of Boston was suffering from syphilis, and I would be the laughing stock of the town. I am going to discontinue the test as worthless': quoted in Dennie, 1962, pp 97-8.

syphilitic organs ... [Wassermann and his team] were searching for their own 'India'... but they unexpectedly discovered a new 'America'...'[32] The negotiations that Fleck described extended to the assessment of individual test results: interpretation, he stressed, was more a mysterious art than a science, and required a delicate initiation passed from one laboratory worker to another. The field of laboratory syphilology was 'a little world of its own ... the serological touch ... more important than calculation ... It is possible to obtain a positive Wassermann reaction from a normal blood sample, and a negative one from a syphilitic sample without any major technical errors'.[33]

Fleck used the test to illustrate his broader philosophy of scientific discovery; and in recent years the book has been admired as perhaps the earliest account of a sociology of scientific knowledge.[34] I am interested less in the detailed development of the test than in its reception and application; nevertheless, Fleck's themes were clearly well appreciated by contemporary practitioners, and formed the basis of intense arguments about the tool's reliability and, by extension, about the place of the laboratory in medical practice. First, the performance of this 'magnificent fluke' was extremely complex - as can be appreciated by reading any description in a contemporary manual.[35] It was quickly recognised that the procedure would have to be based firmly in the laboratory and performed by an expert; this was not an activity for the ordinary practitioner to carry out in his ward side-room - although during the

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[32] Ibid, p 69. For later theories of the test's mechanism, see A M Silverstein, A History of Immunology (San Diego, Ca: Academic Press, 1989), p 311; D J Bibel, Milestones in Immunology (Madison: Science Tech Publishers, 1988), pp 269 - 270.
[33] Fleck, 1935, p 53.

early days attempts to simplify the test were partly driven by this desire. But the BMJ quickly asserted the importance of keeping it in the hands of laboratory workers: 'The Wassermann serum test ... can only be attained in a well-equipped laboratory by one well-versed in laboratory methods. We deplore the attempts to simplify the procedure so as to adapt it for use as a clinical test ...'[36] The dermatologist McDonagh, in his practical guide to using the test, distanced himself from its rarefied complexity: 'It is unnecessary for a busy medical man to burden himself with the theory of a test which can only be carried out in a well-equipped laboratory, with a menagerie attached. However the reaction may be simplified, it can never form a sport for the consulting-room ...'[37]

This complexity, together with its unexplained workings, heightened the perception of a rather mysterious procedure; and clinicians were well aware of the apparently arbitrary way in which a positive or negative result was decided. Contemporary literature often discussed the paradox that, whilst the test was developed in order to diagnose a clinical state, this very same clinical state was used to test the validity of the reaction; thus it was accepted that in up to 30% of cases of secondary syphilis, the obvious clinical state proved the test to be falsely negative! This point was demonstrated in a 1919 study, in which doctors tried to assess its accuracy by testing it against various other criteria of syphilis: clinical signs;

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[34] His claim: 'In science, just as in art and in life, only that which is true to culture is true to nature ...' is original for the period: Ibid, p 35.
[35] The Wassermann reaction was described as such in 'Science Notes', BMJ, Jan 10, 1914, p 101.
[36] Leader, BMJ, 1911, p 692.
[37] J E R McDonagh, 'Wassermann's reaction from a practical point of view', Lancet, April 2, 1910, pp 920-2; on p 920.

pathology; history, and - bafflingly - a previous positive Wassermann result.[38] The circle of evidence was, it seems, difficult to escape. The ambivalence expressed by the physician Henry Morris during the early days of the test was typical of many doctors: 'With such uncertain, variable, and paradoxical results as I have quoted, are we really in a position at present to say what the precise meaning and value of the Wassermann reaction is?' Describing an initiation into the wonders of the laboratory, he wrote ironically:

'It all seemed to me very complicated and very marvellous, and I wondered how it had ever entered into the thoughts of man to apply rabbit's serum and guinea-pig's serum to sheep's corpuscles, then to add an extract of the liver of a congenital syphilitic infant (or of a liver which is not from a syphilitic subject ...) and mix up with them all some of the serum of a patient whose blood is to be tested, and then to draw a momentous conclusion based on whether the red blood corpuscles of the patient sink to the bottom of the test-tube or break up and yield their colouring matter to the whole volume of the test-tube's contents ... Of this I feel sure, that only very competent and well-practised workers are fit to be entrusted with these investigations. And what do we learn from them? Why this: That the experts have arrived at no unanimous opinion as to the precise significance and value of the Wassermann reaction ..!'[39]

Many practitioners, then, felt themselves well and truly in the hands of laboratory scientists, and despite numerous modifications of the test in the years after

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[38] Fildes and Parnell, 'MRC Special Report Series No 23: An Analysis of the Results of Wassermann Reactions in 1,435 cases of Suspected Syphilis', 1919. PRO, Kew: FD4/23.
[39] Henry Morris, 'Observations on Syphilis', Lancet, Aug 4, 1912, pp 498 - 506; on pp 502-3.

its introduction, confusion reigned.[40] Time and again doctors complained that inconsistencies between different laboratories, and even within the same laboratory at different times, were rife. An MRC enquiry, published in 1918, acknowledged that it was impossible to lay down a standard procedure, since 'admittedly the various factors in the reaction, and the proportions in which the reagents afford the best results, have so far been selected only by individual experience and predilection ...'[41] As frustrations continued, attention focused increasingly upon the figure of the pathologist performing the test. On the one hand, aspersions could be cast upon his credibility - as Morris demonstrated. On the other hand, he could be presented as the epitome of expertise, whose personal reputation alone gave the test its authority. The 1918 MRC report, whilst admitting that there were problems inherent in its design, maintained that it was ultimately the laboratory scientist who held responsibility for the test's reliability: 'There is no process of biochemical diagnosis that gives more trustworthy information ... than the Wassermann test when it is performed with completeness and with proper skill and care ... The responsibility attaching to any imperfection or slovenliness of technique is so great, and the results of a false diagnosis may bear so heavily upon a patient and his family, that the Committee believe that the whole weight of responsibility for the reports made should be borne by a fully-qualified pathologist, and that in no circumstances should the reputation of the pathologist in charge be used as a cover for the work of an assistant, however long

[40] For an example of the problems involved in modification, see L W Cann and S De Navasquez, 'The Relative Values of the Kahn and Wassermann Reaction based upon a Survey of 175 Clinical Cases and Tests upon 5000 sera', Brit J Ven Dis, 1931, Vol 7, No 2, pp 105 - 119.

[41] 'MRC Special Report Series No 14: Reports of the Special Committee upon the Standardisation of Pathological Methods: The Wassermann test (interim report)', 1918. PRO, Kew: FD4/14, p 6.

his experience or however great his manipulative skill ...'[42] The American venereologist Stokes, in his widely read 1926 text, took this question of personal integrity to extremes. In his brilliant description of the delicate process - sensitive to many 'intangible elements' - by which the test was interpreted, he warned: 'There are few laboratory procedures whose inevitable margin of error entails more social and personal suffering than the Wassermann reaction. Whatever savours of technical inexperience, of the injudicious and uncritical temperament, of personal motive and purely commercial interest, of haste, inaccuracy, impressionism, and pig-headedness has no place in its performance.'[43]

Attempts to standardise moved to an international level during the early 1920's, culminating in a series of recommendations issued by the League of Nations.[44] By now the Wassermann had been replaced in many countries by newer 'flocculation' tests, but British laboratories still tended to use the old method. By the 1930's, exasperation was still common: as two psychiatrists commented in 1933: 'The technique of the Wassermann is cumbersome, almost out of reach of smaller laboratories in which there is no full-time pathologist to supervise, poorly sensitive, and not particularly specific ... One wonders at the desperate clinging to the Wassermann that characterises British syphilology ...'[45]

Technical suspicions about the test were extended to more general concerns that the laboratory might be usurping the rôle of clinical experience: medicine,

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[42] Ibid, p 21.
[43] J H Stokes, Modern Clinical Syphilology (Philadelphia and London: WB Saunders Co, 1926), p 80.
[44] See report: 'International conference on the standardisation of sera and serological tests', Lancet, Dec 9, 1922, pp 1238-40.

clinicians constantly stressed, was an art as well as a science. Shortly after its introduction there was a clear current of suspicion towards what it symbolised, exemplified by the St Bartholomew surgeon D'Arcy Power's comments. Describing his early resentment towards the surrender of clinical authority, he confessed: '[I found it] difficult to believe that a young man, seemingly in perfect health and without a blemish, was suffering from a spirochaete infection simply on a pathological report ... I knew and had taught for years that syphilis was a deceitful disease ... but it was difficult to rely upon a test made by another person, however skilful and assured he might be ...'[46] He went on to concede that now, several years later, he had reluctant faith in the test and must rely upon the skill of the laboratory expert - however 'repugnant to my surgical instinct.'[47] Such suspicion of 'paper syphilis' - and an eagerness to reassert clinical values - was common amongst doctors in all areas of medicine.[48] The neurologist Buzzard, whilst acknowledging the value of the test, commented that '... The ordinary physician like himself ... needed to give an opinion without constant resort to laboratory tests ... In view of the test-tube tendencies of the present time he did not think one could urge too strongly the importance of teaching medical students all there was to know about ordinary bed-side methods of examination...'[49] The army surgeon French similarly cautioned against the allure of the laboratory: 'I put the clinical aspect [of diagnosis] last, but in my opinion it is by no means the least valuable. It does not perhaps glitter like the

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[45] J E Nicole and E J Fitzgerald, 'The Sero-Diagnosis of Syphilis in Mental Hospital Practice', J Ment Sci, 1933, 79, pp 52 - 88; on p 87, p 86.
[46] D'Arcy Power, 'On the Treatment of Syphilis', BMJ, June 22, 1912, pp 1418-21; on p 1418.
[47] Ibid, p 1418.
[48] The term - referring to syphilis diagnosed simply on the basis of a test report - was coined by Stokes.

gold of the spirochaete, nor sound like the brazen cymbal of a positive Wassermann reaction, but as regards induration in the chancre it has the intrinsic merit of home manufacture ... Whatever each individual may think about the relative significance of Wassermann reactions, the fact must be admitted that in many instances we are as dependant today upon a correct clinical interpretation of what we see as in the past when John Hunter wrote.'[50]

Contrary to expectation, such undercurrents of antagonism increased after the War. Technical confusion, as I have described, was increasing; and many now claimed that the test had been accepted too readily in the early days, had become over-valued, and was leading either to dangerously over-enthusiastic treatment, or to neglect of treatment if too much faith was placed in negative results.[51] In 1921 the BMJ aired some views by no means uncommon amongst venereologists. Bayly noted that the unreliability of the test was now 'generally acknowledged', and that there was 'a dangerous tendency at the present day to exalt the value of laboratory diagnosis and neglect that of clinical experience ... During the late war complete reliance was placed by the RAMC authorities in the positive Wassermann reaction, and from personal experience in army Venereal Disease hospitals, I am convinced that very many men were labelled 'Venereal Diseases' who were never infected by the Spirochaete pallida

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[49] 'Discussion by the Medical Society of London of Mott's Paper: Differential Diagnosis of Syphilis and Para-Syphilis of the Nervous System', Lancet, Nov 11, 1911, pp 1480-2.
[50] H C French, 'Hunterian Lectures: Recent developments in the recognition, treatment, and prophylaxis of syphilis', Lancet, Nov 11, 1911, pp 1315-18; on pp 1317-18.
[51] Note Buzzard's comment: 'A negative Wassermann reaction is certainly not evidence of a cure, and the fact that it has been regarded as such has made it an obstacle in the way of efficient treatment since a negative reaction gives rise to delusions of security ... leading to suspension of treatment.' Meeting of the Section of Neurology of the Royal Society of Medicine, Mar 8, 1923: 'The Treatment of Neurosyphilis', Brain, 1923, 46, pp 138-42; on p 138. Dennie, in his traditional history of syphilis,

...'[52] His colleague Thursfield, responding to the article, agreed that patients had to be 'persuaded out of that false, dangerous belief' in the test. Laboratory workers, he claimed, were influenced by clinicians' expectations of results: 'No laboratory worker who knows what is expected of him can enter upon the performance of a Wassermann test without some degree of obsession which to that degree, must impair his strict impartiality and degrade the value of his judgement.' He concluded grandly: 'Laboratory dogma is not necessary truth, and the acceptance of it as such is an instance of the worship of the Idols of the Theatre ...'[53]

Stokes once again elegantly expressed a feeling that clinicians should fight back against the dictatorship of the laboratory: 'It will perhaps be a little difficult for the serologist, accustomed as he has been to the unquestioned acceptance of his dicta by a thoroughly Wassermannized profession to respond with entire good grace to the questionings and criticism of his syphilogic confrère ...'[54] Despite referring to the American situation, his depiction of 'taut rivalry' seemed particularly relevant in Britain during the 1920's, as doctors debated the relative merits of centralised, anonymous laboratories, and local laboratories which might foster co-operation between clinicians and pathologists.[55] Small-scale pathologists, as well as practitioners, were keen to extol the virtues of the friendly co-operative local

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noted: 'Because of the inbred worship of the serological tests when positive, many non-syphilitic patients were treated intensely ... sometimes with disastrous results ...': Dennie, 1962, p 99.

[52] H W Bayly, 'The Value of the Wassermann Reaction', BMJ, May 7, 1921, p 686.

[53] H Thursfield, Letter: 'The Value of the Wassermann reaction', BMJ, May 14, 1921, p 719. For further examples of such copious comments, see K Dickson, 'The Value of the Wassermann Reaction', BMJ, April 30, 1921, p 658; C F Marshall and A G Shera, 'The Treatment of Cases of Syphilis having a Persistent Positive Wassermann Reaction', Lancet, June 18, 1921, p 1299.

[54] Stokes, 1926, p 81.

[55] Ibid, p 32. For a taste of the debates, see the series of letters between 'Clinical Pathologist' and 'Bacteriologist', BMJ, Oct 18, 1919, p 510; Nov 15, 1919, p 649; Nov 29, 1919, p 724.

laboratory: as one happily remarked: '[A country practitioner] recently motored many miles to me with a cerebro-spinal fluid in a sterile bottle snuggled in cotton-wool close to his skin under his waistcoat. That is the spirit I am out to inspire. I would have gone to him and saved him the trouble if he had asked me to.'[56] This situation was contrasted with the 'penny-in-the-slot' system, as ' ... when a specimen from an obscure case is sent to a central laboratory and is examined at best by someone who knows nothing of the case, or worse, by a laboratory assistant who may be good, bad, or indifferent ...'[57]

Ever since the introduction of the test, there had been those who deplored such separation and mutual suspicion between clinical and laboratory experts.[58] During the 1920's, however, the need for co-operation became central to discussions of its reliability. The venereologist Colonel Harrison was a major spokesman for this: clinicians, he claimed, criticised the test because they didn't understand it; pathologists, on the other hand, worked in blinkered isolation, and took no interest in the concerns of the clinician: 'Between the plainly syphilitic reaction and the clearly negative there is a zone into which some non-syphilitic sera now and then wander ... Progress towards higher standards of diagnosis and treatment will not be made where the pathologist and the clinician are not in the closest possible sympathy ...'[59] Here, finally, were two central paradoxes of the Wassermann test. Greeted as the first

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[56] Letter from'Bacteriologist', BMJ, Nov 15, 1919, p 649; see also Letter from H Miller: 'Local Clinical Laboratories', BMJ, Aug 16, 1919, p 22.
[57] Letter, A G Shera, BMJ, Aug 30 1919, pp 287-8; see also Letter, J Ritchie, BMJ, Sept 13, 1919, pp 360-1.
[58] See L W Harrison, 'The Role of the Pathologist in the Recognition and Treatment of Syphilis', BMJ, Sept 23, 1911, pp 686-7; Idem, 'The Need for Co-ordination of Clinical and Laboratory Work', BMJ, Feb 3, 1912, pp 255 - 257.

objective indicator of the elusive disease syphilis, twenty years later it seemed that it could be interpreted only through the intuition of the pathologist, and through close negotiation between the pathologist and the clinician. Calls for standardisation of the test, however, were inimical to this very ideal of localised co-operation.

The Test Enters Psychiatry: Neurosyphilis confirmed

Crichton-Browne's laboratory at the West Riding asylum had provided a model for applying science to psychiatry during the later nineteenth century. In 1895 the London County Council laboratory at Claybury followed this precedent, and would again exploit research in up-to-the-minute fields such as neurophysiology, genetics, and endocrinology. A year later, Thomas Clouston organised a similar enterprise for Scotland - founding the Central Pathology Laboratory of Scottish Asylums, housed at the Royal Edinburgh Asylum under the directorship of the existing asylum pathologist William Ford Robertson. Ford Robertson's major aims were to carry out and promote research into all areas of the pathology of insanity, and to offer laboratory instruction to Scottish asylum medical officers. In 1902, however, he tried to direct the laboratory specifically into the new paths of bacteriology; calling, in particular, for greater emphasis upon the doctrine of immunity as it might apply to GPI.

Robertson rejected the theory that syphilis played a prime role in the disease, and his main arguments were clinical - particularly the objection that few syphilitics went on to become general paralytics. His alternative, however, sprang from the conviction that the key to curing GPI lay in serum and vaccine therapy. He developed

[59] Col L W Harrison, 'Presidential Address: The Value of a Scientific Outlook to the Worker in

the hypothesis that the disease was caused by diphtherioid bacilli which colonised the gut, respiratory tract, and blood-stream - much as a handful of researchers were suggesting that tabes was caused by septic foci in the urinary tract. The role of syphilis, he argued, was simply to weaken the defences, allowing such infection to take hold.[60] Clouston was in support, agreeing that his colleague had 'proved that the immediate cause of the disease was a microbe which acted specially on brains that had previously been weakened by dissipation and poisoning ...'[61] Robertson's work was a perfect example of the desire to apply the new laboratory ethos to psychiatry. He supported his hypothesis with extensive evidence from bacteriological culture and animal experimentation, and by 1908 was announcing that anti-sera to the diphtherioid toxin was effective in arresting GPI. Syphilis, resistant for so long to microbiological tools, was placed in the background - remaining the rather vague 'diathesis' of the nineteenth century. The diphtherioid bacillus, which could be grown, visualised, and transferred between species, was placed in the foreground - as the immediate cause of GPI. The Lancet cautiously encouraged the Scottish work; however, by 1908 the newly developed Wassermann test was promising to offer syphilis the concrete laboratory status it had lacked.[62] Although Ford Robertson was still challenging the role of syphilis as late as 1915, his work faded into the background; and perhaps disappointed by his lack of success, he subsequently turned his attention to bacterial infection and therapeutic immunisation in dementia praecox.

Venereal diseases', Brit J Ven Dis, 1925, vol 1, no 2, pp 81 - 85; on p 82.

[60] W Ford Robertson, 'The Morison Lectures for 1906: The Pathology of GPI', J Ment Sci, 1906, 52, pp 278 - 284.

[61] T Clouston, 'Annual Report of the Royal Edinburgh Asylum', Lancet, Mar 10, 1906, pp 714-5.

[62] See Leader 'The Bacillus Paralyticans and GPI', Lancet, July 6, 1907, pp 33-4.

This short episode illustrates the increasing allure of laboratory medicine, and more or less fluid conceptions of germ theory, to psychiatrists at the beginning of the twentieth century.[63] But as Ford Robertson seemed to pursue a red herring, the majority of his colleagues were caught up in the exciting possibilities of the new Wassermann test. One application of the technique was immediately obvious: to test the aetiological rôle of syphilis in GPI. By coupling their technique with the relatively new clinical procedure of lumbar puncture, Wassermann and a co-worker Plaut had demonstrated in their first experiments that the syphilitic 'anti-stoff' was present in roughly 90% of samples of both serum and cerebro-spinal fluid from clinically diagnosed general paralytics. From 1908, a multitude of figures were published in Britain which corroborated these results; and by 1917, the quoted percentages had risen to between 98 and 100% positive - far higher, in fact, than were yielded in any stage of syphilis.[64] Since in psychiatric circles it was almost immediately accepted that the Wassermann test was an accurate indicator of syphilis, these observations naturally confirmed the disease's aetiological role in GPI - increasing by 15 to 20 per cent the accepted proportions of syphilitic antecedents averaged from statistical evidence alone. As an early article in the Journal of Mental Science concluded: 'If then it be allowed ... that a positive Wassermann means

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[63] See Bynum, in Berrios and Freeman, 1991, p 172; A Scull, 'Desperate Remedies: A Gothic Tale of Madness and Modern Medicine', Psychological Medicine, 1979, 17, pp 516 - 577.

[64] For a summary of early results, see H A Schölberg and E Goodall, 'On the Wassermann reaction in 172 cases of mental disorder (Cardiff City Mental Hospital) and 66 control cases, syphilitic and other (chiefly from Cardiff Infirmary), with historical survey for the years 1906 - 10 inclusive: Comments and conclusions', J Ment Sci, 1911, 57, pp 218 - 273; also C H Browning and I McKenzie, 'On the Wassermann reaction, and especially its significance in relation to general paralysis', J Ment Sci, 1909, 55, pp 437 - 447; p 439.

syphilis, we have the fact established that general paralysis is an affection almost invariably associated with syphilis'.[65]

Assessing the impact of the test upon perceptions of GPI is not easy; and highlights the conflicting evidence that we sometimes find when examining books and journals on the one hand, and practice as shown by clinical records on the other. Early twentieth-century doctors already allowed syphilis a prime place within a multifactorial framework of GPI, and appealed quite naturally to 'syphilisation and civilisation'. Rates of change in individual asylum observations, however, differed widely. At Hanwell Asylum, for example, there was a dramatic rise in the percentage of cases assigned to syphilis between 1890 and 1900.[66] At Claybury, in contrast, this change did not take place until between 1910 and 1915, and was a direct result of using the Wassermann test. It seems unlikely that these differences were due to differences in knowledge of current theories; or that psychiatrists at Claybury - the territory of Mott - were in disagreement with the theory of syphilitic origin before the 1910's. More likely is that psychiatrists here were reluctant to assign syphilis as a cause in the absence of a clear history or signs in individual patients; with limited time, it was far simpler to fall back upon the old standards of alcoholism or heredity.

Claybury asylum was opened in 1893; and whilst fairly typical of a large late-Victorian institution, it was unusual in that it was also the site, between 1895 and 1914, of the Central Laboratory, under Mott's direction.[67] The average number of

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[65] G Scott Williamson, 'The cerebro-spinal fluid in general paralysis and the nervous lues', J Ment Sci, 1909, 55, pp 655 - 665; on p 660.
[66] From 2% of cases in 1890 to 38% in 1900: Male clinical case-books of Hanwell Asylum: 1890 (GLRO: H11/HLL/B20/20); 1900 (B20/28 and 29).
[67] For an account of the asylum, see Pryor, 1993.

annual admissions between 1900 and 1916 was 230; and of these an average of 16% received an initial diagnosis of GPI.[68] The Wassermann test was first introduced at the asylum during the middle of 1909; during 1910 it was performed upon 4% of the total admissions; and by 1912 this figure had risen rapidly to 26%, a proportion which remained fairly constant over the next ten years. When the test was performed, both blood samples and cerebro-spinal fluid samples were always taken. Immediately prior to its use, the commonest assigned causes for general paralytics were still mental stress, alcohol, shock, injury and heredity. Syphilis was occasionally implicated if the patient gave a clear history, or displayed clinical signs of the disease. As the Wassermann test was increasingly used - and practically always found to be positive in those patients with an initial diagnosis of GPI - syphilis was increasingly entered as the sole cause. Thus during 1910, syphilis constituted 22% of all assigned causes of GPI; by 1912 it had risen to 61%; by 1916, 80%. The remainder of the causes - attributed to those patients who were not tested or who (rarely) gave a negative reaction despite clear symptoms of GPI - were divided between the traditional factors. The change demonstrates how, in the local context, this new piece of laboratory technology was quickly accepted; and, as a reliable 'lie-detector', confirmed the GPI-syphilis link in a far more concrete and impressive way than could statistical studies.

Despite its power, the test could not frame fully scientifically the nature of the link between syphilis and parasyphilis - described by Fournier's rather obscure phrase: 'cause not provenance'. Wassermann had presumed that a positive test

[68] Male and female clinical case-books of Claybury Asylum (1900 to 1916). These documents were formerly kept in the archives of Claybury Hospital. Unfortunately they were destroyed in 1995,

indicated active syphilis - that is the active presence of the spirochaete; however, as questions were raised about the theoretical basis of the reaction, it became doubtful whether this was the case. Carl Hamilton Browning and Ivy McKenzie, microbiologist and physician respectively, published a number of papers upon the application of the Wassermann test, based upon their work at the Pathological Laboratory of Glasgow University and the Western Infirmary. They commented in 1909: 'It is as yet impossible to say whether or not the fact that a serum yields a positive result is a proof of the presence and pathogenic activity of living organisms in the host.'[69] As long as the test only indicated the secondary effects of infection, they pointed out, GPI could still be regarded as a disease with an indirect link - perhaps via a toxic product.[70] Their subsequent arguments against an active 'contagium vivum' in GPI - which included the resistance of the disease to anti-syphilitics - revealed that the old problems of definition had not been entirely abolished by the laboratory.

It took a second piece of triumphant science to settle this question with more satisfaction. Increasingly, laboratory culture demanded that organisms actually be visualised within pathological lesions in order to prove aetiology: but the spirochaete had long eluded observers of parasyphilis - although it had been found in strictly tertiary lesions such as aortitis. In 1912 Hideyo Noguchi and JW Moore, two scientists working at the New York Rockefeller Institute, finally demonstrated the

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although a small number of the better-preserved books were kept, with plans to remove them to a local library at a later date.

[69] Browning and McKenzie, 1909, p 440.

spirochaete in the post-mortem brains of twelve out of seventy cases of general paralysis.[71] Their report - and a relevant section of brain - was quickly sent to the Glasgow University Laboratory, whence the news was passed on to the British medical community. In the United States, this particular piece of work was a source of great national pride in the organisation of science: 'Noguchi and Moore's work ... indicate to us as Americans what the establishment of scientific institutes may do to permit the rapid application of new ideas to branches of enquiry that are opened out.'[72] It was also regarded as a potential way of aligning psychiatry with the higher-status speciality of neurology: 'The net result of the modern work on neurosyphilis has been to bring the neurologist and the psychiatrist together upon one platform in diagnosis and more and more upon one platform in treatment.'[73]

To British contemporaries, also lured by the possibilities of the laboratory, the work was indeed an 'epoch-making discovery'.[74] The organism had, of course, been found in only a small sample proportion, and there were occasional suggestions that GPI should be defined strictly according to whether spirochaetes were visible in individual post-mortem brains.[75] This, however, was seen as both impractical and irrelevant beside the fact that a concrete microbiological breakthrough had been made. Mott, who confirmed Noguchi and Moore's work at the Claybury Laboratory

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[70] See, for example, Strumpnell and Möbius' theory of syphilitic toxins, referred to in W W Ireland, 'Review of the Statistics, Aetiology, Symptoms and Pathology of General Paralysis by Junius P and Arndt M', J Ment Sci, 1909, 55, p 536.
[71] H Noguchi and J W Moore, 'A Demonstration of Treponema Pallidum in the Brain in Cases of General Paresis', J Exp Med, Feb 1913, 17, pp 232 - 238.
[72] E E Southard and H C Solomon, Neurosyphilis: Modern Systematic Diagnosis and Treatment: Presented in One Hundred and Thirty-Seven Case Histories (Boston: W M Leonard, 1917), p 427.
[73] Southard and Solomon, 1917, p 432.
[74] Robertson, 1913, p 211.

concluded: 'We have a new conception of the disease on the sure ground of direct observation ...'[76] The visualisation of the spirochaete now outweighed the objection that GPI pathology was not typical syphilitic pathology: 'The problem of the aetiology of general paralysis', wrote the Glasgow psychiatrist George Robertson (not to be confused with Ford), 'appears to have been finally and conclusively settled by the finding ... The theories of a parasyphilitic toxin, of secondary infections or of other accessory factors, sink into insignificance beside this convincing fact ... general paralysis is a manifestation of active syphilis ... The keystone has now been found and fitted to the arch, completing a solid structure on which we can safely base our theories of prophylaxis and treatment.'[77] The Lancet agreed: 'The importance of the announcement can scarcely be exaggerated, and it is probable that we are on the eve of still more significant discoveries. No one can doubt that the ultimate result of such researches will be to engender a ray of hope, from a therapeutic standpoint, where at present the physician can see little to encourage him'; 'Such facts ... are calculated to revolutionise the accepted opinions on the nature of parasyphilis.'[78]

Laboratory medicine indeed seemed to have completed the reformulation of syphilis: curative criteria were no longer relevant, and clinical criteria no longer paramount. 'Parasyphilis' was quickly dropped from the medical vocabulary, to be replaced by the new term 'neurosyphilis' - unequivocal syphilis of the nervous system manifesting as GPI or tabes dorsalis. Of course the old clinical problems remained:

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[75] See, for example, M Robinson, 'Some doubtful cases of so-called GPI', J Ment Sci, 1914, 60, pp 291 - 295.
[76] F W Mott, 'The Degeneration of the Neurone in the Light of Recent Research', Lancet, Nov 15, 1913, pp 1367 - 1375; on p 1368.
[77] Robertson, 1913, p 211.

why only a few syphilitics developed the disease, and why uncivilised countries in which syphilis was rife had relatively little GPI. For these reasons factors besides syphilis would continue to find their way into causal explanations. However, the central causal importance of syphilis was now accepted, and during the first decade such multifactorial arguments would come to be framed increasingly in terms of microbiological and immunological theory.[79]

Historians have observed that, notwithstanding medical advances in the management of syphilis during the first part of the twentieth century, moral attitudes towards the disease changed little: 'venereal disease remained a symptom of social decay and sexual evil.'[80] On the evidence of published writings, the pejorative associations of syphilis had clearly come to apply to GPI over the turn of the century. The attitudes of individual asylum doctors, however, are much more difficult to glean. Braslow draws upon a rich clinical archive from the American Patton State Hospital, which allows him to suggest that early twentieth-century doctors characterised their syphilitic patients in pejorative ways.[81] It is open to question whether terms such as 'lazy', 'dirty', or 'immoral' applied particularly to syphilitics, as Braslow suggests. In the British archives that I have consulted, such admonitory tones were used by doctors to refer to the difficult behaviour of any of their asylum charges. In addition the notes are far more terse, and do not allow one to draw firm conclusions about their moral stance. Between 1900 and 1905, Claybury doctors typically noted 'intemperance',

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[78] Leader, Lancet, Mar 29, 1913, p 911; 'New Light on the Problem of Parasyphilis', Lancet, Aug 23, 1913, pp 577-8.
[79] I return to this theme in Chapter 5 below.
[80] Brandt, 1987, p 160.
[81] Braslow, 1996.

'past gay life', and 'dissipated habits' in general paralytics; but they were equally likely to note 'worry', 'brilliance', 'industry', and 'steadiness'.[82] By the years 1910 to 1915 the most noticeable changes were the frequency of the unembellished phrase 'had syphilis in the past'; and the new emphasis upon the lumbar puncture and the Wassermann test results. These demonstrate above all that the patient's disease was increasingly regarded in the light of the laboratory. Although we might speculate that, with the firm aetiological link, the patient was also increasingly morally censured, this can be no more than a speculation.

The Wassermann Test Diagnoses GPI

References to the Wassermann test in the British literature did not dwell solely upon its importance in proving the syphilitic aetiology of GPI; instead psychiatrists quickly turned to the possibilities that it offered as a diagnostic tool for the disease. The speed with which the test became accepted as such suggests that a positive result in a suggestive patient (whether from the blood or the cerebro-spinal fluid) quickly became practically synonymous with a diagnosis of neurosyphilis. Soon after its introduction, psychiatrists were recommending that all suspected cases of GPI should submit to both vein and lumbar puncture; and for many who published in the early years of the test, the new 'Wassermann' definition of GPI seemed triumphant over the old clinical criteria. In 1912 the Lancet noted a consensus that: ' ... a positive Wassermann reaction is conclusive evidence of the presence of disease even in the

[82] Clinical case-books of Claybury Asylum.

total absence of all manifestations.'[83] George Robertson confirmed: 'If the Wassermann reaction be negative in the spinal fluid as well as in the blood-serum then general paralysis may now, with almost absolute certainty, be excluded in spite of clinical symptoms.'[84] A year later he was fulsome in his praise of the laboratory revolution: 'As a consequence of the discovery of these new reactions and signs we have attained to an accuracy in the diagnosis of general paralysis unapproached in the past, and not excelled in the case of any other disease as important. There are few departments of clinical medicine in which, during the last 10 years, more valuable additions to our knowledge have been made'[85]

Such triumphant rhetoric - in which psychiatrists linked laboratory advances to the desired progress of their specialty - was common in the years leading up to the War, and psychiatrists seemed to express far less antagonism or suspicion towards the test than did their general medical colleagues. The Journal of Mental Science typically hailed the 'triumph of applying biological, chemical and physical sciences to study the causes of mental disease', and confidently predicted that advances in GPI would lead the way for successes in all areas of mental medicine.[86] The episode was used as a weapon in the battle between mainstream psychiatrists advocating the medical model of mental disease, and those following psychoanalytical approaches. Thomson's presidential address to the Medico-Psychological Association in 1914 stressed the importance of science in advancing his specialty, citing the application of

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[83] 'Our Knowledge of Syphilis', 1912, pp 163-4. My italics.
[84] Robertson, 1913, p 196. My italics.
[85] G Robertson, 'The Serum and CSF Reactions as Signs of General Paralysis', J Ment Sci, 1914, 60, pp 1 - 17; on p 17.
[86] 'Asylum reports', J Ment Sci, 1915, 61, pp 299 - 302 ; p 300.

germ theory to GPI as a prime example: 'What hopeful vistas of increasing knowledge and power do such discoveries hold out to us compared to the barren psychological and metaphysical theorems of half a century ago, some of whose ghosts in brand new shrouds are being enticed back to life in quite recent years.'[87] Ernest Jones, champion of these 'newly-shrouded ghosts', certainly regarded the work upon GPI as a force to be reckoned with. In the year that the Wassermann test became widely used in Britain, he spoke with regret of the new discoveries relating to neurosyphilis - in a way which foreshadowed arguments put forward by Thomas Szasz sixty years later. Having observed that GPI could now be delineated as a sharply-defined disease whose aetiology was understood, he warned: 'I am greatly impressed with the danger there is in tending to regard general paralysis as a paradigma of mental disease. This tendency is an extremely natural one, because it is so harmoniously in accord with the over-materialistic training we have all received ...' But GPI would not, he insisted, provide the hoped-for breakthrough in psychiatry: 'Some of us ... are tempted at times to regret the making of these discoveries, magnificent as they are. At all events, I feel sure that they will set back the clock in the development of scientific psychiatry more thoroughly than any other event that could have happened ...'[88]

Such rhetoric masked the complexities surrounding the practical application and impact of the test. It is not easy to gauge how quickly or comprehensively it was

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[87] DG Thomson, 'The Presidential Address on the Progress of Psychiatry during the Past Hundred Years, together with the History of the Norfolk County Asylum during the same Period', J Ment Sci, 1914, 50, pp 541 - 572.

[88] E Jones, 'Modern Progress in our Knowledge of the Pathology of General Paralysis', Lancet, July 24, 1909, pp 209 - 212; on p 211.

incorporated into asylum practice, since published reports and clinical records offer only oblique references to laboratory work. Between 1910 and 1925, there was a steady increase in both the number of asylums boasting on-site laboratory facilities, and the number of Wassermann tests performed upon in-patients. The Commissioners, and their successors the Board of Control, continually exhorted asylums to develop their own small laboratories, 'instead of relying on the present inadequate method of posting a selection of specimens for examination elsewhere...'[89] By 1924, they reported that 60% of asylums had laboratories in more or less active operation: about half of these had a trained laboratory assistant, and fourteen housed a resident pathologist. The figure had risen to 80% by the end of the decade. In parallel, the Board noted a steady advance in the number of asylums and 'institutions for defectives' in which new admissions routinely had blood Wassermann tests taken. In 1928, a survey revealed that 30% of the 96 British public asylums claimed to be testing all of their admissions, or all of their male admissions. The remaining asylums tested those in whom a diagnosis of GPI was suspected; and CSF tests were generally only performed in those yielding a positive blood result.[90] The Board urged that the Wassermann should become a uniform procedure - and suggested that even routine lumbar punctures might be desirable.[91] It seems probable, then, that the Wassermann test provided a great stimulus to the development of pathological laboratories associated with asylums - a point which psychiatrists naturally saw as extremely

[89] Eleventh Annual Report of the Board of Control for 1924 (London: HMSO, 1925), p 18.
[90] Correspondence of the Board of Control, 1928. PRO, Kew: MH 51/ 698, Part 2.
[91] Seventeenth Annual Report of the Board of Control for 1930 (London: HMSO, 1931), p 99.

beneficial.[92] It also seems that, in contrast to the situation in general medicine, psychiatrists were surer of the benefits of performing their own tests on site, rather than sending them away to larger, more standardised laboratories.

But although the test quickly became a mainstay of asylum practice, we have to be cautious in suggesting that it transformed GPI from a symptom-defined to a laboratory-defined disease.[93] We cannot assume, for example, that it really led to great changes in either the numbers of general paralytics diagnosed within asylum walls, or the way in which the diagnosis was made. Hare, in his detailed epidemiological study of the disorder, noted: 'I cannot find that, on the whole, there was any sudden fluctuation in the reported incidence or mortality of the disease after the introduction of objective methods of diagnosis...' - a statement which supported his own suggestion that GPI had been accurately diagnosed during the nineteenth century, and so was amenable to retrospective analysis.[94] The Registrar General's mortality figures for GPI showed a steady decline from 1901 onwards, with no short-term fluctuation in the years surrounding the introduction of the test. Their use in this context, however, is dubious: it is probable that they would have been gleaned primarily from post-mortem and clinical criteria, with less direct relationship to Wassermann diagnostic practice. More relevant are the figures for annual first admissions to asylums, reported by the Commissioners and, from 1914, by the Board of Control. These showed a steady rise between 1900 and 1912 to 1700, followed by a sudden drop in 1914, which contemporaries attributed to the outbreak of the War.

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[92] See T D Power, 'The Aetiology of GPI', J Ment Sci, 1930, 76, pp 524 - 536; p 529.
[93] As, for example, Cunningham portrays the transformation of plague at the beginning of the twentieth century: A Cunningham, 'Transforming Plague', in Cunningham and Williams, 1992, pp 209 - 244.

Figures during the War were incomplete; and by the time they had again attained a degree of accuracy in 1920, asylums admissions for GPI had dropped to 1300 - a trend which would continue steadily for the next thirty years.[95]

Such changes are open to a number of interpretations - as I discuss further in Chapter 5 below. It is certainly possible that a proportion of the drop in admissions from 1912 was due to the Wassermann test reducing the number of patients in which the diagnosis was made - but it is unlikely that it was considerable. More importantly, the figures counter the claim that the test might have been used to over-diagnose general paralysis in asylums through an exaggerated faith in laboratory criteria. George Robertson, a self-confessed devotee of the Wassermann test, estimated that it had increased the diagnostic accuracy of GPI by 6 to 15 per cent, primarily by differentiating it from two conditions agreed to present most similarly - cerebral syphilis and alcoholic insanity. Such estimates were based upon studies - difficult to substantiate - in which diagnoses during life were compared with post-mortem diagnoses.[96] But the archives of Claybury asylum support the suspicion - in the local context at least - that older clinical and newer laboratory criteria were not greatly different.

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[94] Hare, 1959, p 613.

[95] Annual Reports of the Commissioners in Lunacy (1900 to 1912); Annual Reports of the Board of Control (1914 to 1920).

[96] Robertson, 1913, p 215. The test was generally claimed to be positive in about 70% of cases of cerebral syphilis, and in 98 to 100% of cases of GPI. Whilst a negative test therefore made GPI very unlikely, a positive test could indicate either disease. Robertson evidently assumed that, of the negative-testing patients (now diagnosed either cerebral syphilis or alcoholic insanity), a proportion might previously have been mistaken for GPI. Similarly, of the positive-testing patients (now diagnosed either GPI or cerebral syphilis according to clinical likelihood), a proportion might previously have been mistaken for alcoholic insanity. He did not specify, however, whether the number of GPI cases diagnosed therefore rose or fell. For figures of Wassermann positivity see Southard and Solomon, 1917, p 82.

Case and hospital notes - although they might offer a more accurate view of medical practice than journals and texts - present their own interpretative problems, and can still only tell us a limited amount about how diagnostic decisions were made.[97] In most of the Claybury notes, for example, it was not documented exactly when the Wassermann test was performed; neither is it easy to tell at which point during their stay patients were assigned to specific diagnostic categories - particularly as many of the entries were copied into the admissions book from earlier notes. Bearing in mind these qualifications, however, it is possible to make some general observations. The Wassermann test was performed only on those patients who were assumed clinically to have GPI on presentation, whose diagnosis was unclear, or who (in a few cases) had a history or signs of syphilis. Between 1910 and 1916, for example, an average of 66% of the tests were performed on patients with an assigned diagnosis of 'general paralysis' or '?general paralysis' on admission; and the remainder on patients with various initial diagnoses such as melancholia, mania, and confusional insanity. Furthermore, a positive or negative test result in itself was rarely sufficient to alter an initial diagnostic category. In the majority of cases the test confirmed the assumption on admission as to whether GPI was present or absent. In those cases where the test was contradictory, the initial diagnostic category was rarely changed; if it was, it was usually documented to have been changed due to a change in clinical features. Such observations suggest that, in this limited context at least, the test itself was not over-enthusiastically used to define the diagnosis of GPI. Despite

[97] For a discussion of these problems see G B Risse and J H Warner, 'Reconstructing Clinical Activities: Patient Records in Medical History', Social History of Medicine, 1992, 5, pp 183 - 205.

rhetoric about the power of the test, many psychiatrists insisted that their clinical skills were aided, not replaced, by this new laboratory tool.[98]

The Wassermann test and Early GPI

Of course once a patient had entered an asylum with suspected GPI, even a more accurate diagnosis would have little effect upon his fate during the early part of the century. Partly for this reason the power of the test appeared to be greatest when it was applied to early or hidden disease in the non-asylum population. Uncovering the relationship between GPI and syphilis had allowed psychiatrists to participate in the general alarm surrounding the pervasiveness of the venereal taint. Numerous articles linked the prevalence of hidden syphilis in the community with the apparently rising numbers of the insane: 'Over twelve hundred men, in the prime of life, most of them strong, useful, vigorous ... fall victims to a rapidly fatal illness, general paralysis, due to a preventable cause, syphilis ...'; 'It is terrible to think from these [asylum] figures that such a large amount of poison dwells unrecognised among us ...'[99] Such writings in the post-laboratory era echoed Fournier's call to arms - stimulated by statistical evidence - of twenty years before. The test, however, appeared to be a far more powerful indicator of the danger; and it allowed doctors to push the idea of the 'prodrome' - the earliest manifestation of neurosyphilis - to its absolute limits.

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[98] 'These newer laboratory methods of course can never replace accurate observation of the well-known physical signs of ...[GPI] ... but a knowledge of them is of the utmost importance when we have to deal with obscure and doubtful cases.': G W Ross and E Jones, 'On the Use of Certain New Chemical Tests in the Diagnosis of General Paralysis and Tabes', BMJ, May 8, 1909, pp 1111-3; also Mott: 'Post-mortem has shown that the Wassermann in combination with clinical signs is an accurate method of diagnosis ...': F W Mott, 'Diagnosis and Treatment of Parenchymatous Syphilis', J Ment Sci, 1915, 61, pp 175 - 197.

[99] Asylum reports, J Ment Sci, 1914, p 135; J Ment Sci, 1915, 16, p 303.

During the 1910's and 1920's, psychiatrists were stepping up frequently-voiced calls for a profession based outside the asylum - in out-patient clinics, general hospital wards and in the community - which would target patients who, whilst mentally ill, were not strictly insane. This project of diversification, they hoped, would separate chronic from acute and early cases, and thus promote psychiatry as a scientific and therapeutic discipline rather than a custodial one. Before 1930, little progress was made. Hospitals and asylums were reluctant to open out-patient clinics, although about ten were running by the early 1920's, and this figure would increase steadily over the next fifteen years.[100] In 1915, the London County Council Act gave the Maudsley Hospital unique powers to admit voluntary patients, and it rapidly became the flagship institution for the ideal of non-asylum treatment.[101] During the early 1920's it received roughly 500 in-patients and 900 out-patients annually, made up of referrals from private doctors, relatives, general hospitals, poor-law wards, educational and charity organisations, and from patients themselves. Finally in 1930, the Mental Treatment Act conferred optional powers on local authorities to admit voluntary patients to mental hospitals, to organise out-patient treatment and after-care, and to fund research into the prevention of mental disease.[102]

Such developments potentially provided a backdrop for effectively applying the Wassermann test to early neurosyphilis. In the United States the work of the Boston Psychopathic Hospital - described by Elizabeth Lunbeck - offered a clear

[100] The earliest of these had opened in 1890 at West Riding asylum and at St Thomas' Hospital.
[101] For an account of the origin of the hospital - and particularly the roles of Mott and Henry Maudsley, see P Allderidge, 'The Foundation of the Maudsley Hospital', in Berrios and Freeman, 1991, pp 79 - 88.

example of how the test might contribute successfully to such a broad professional project for psychiatry.[103] The basis of Lunbeck's thesis is the attempt by the staff of this hospital - particularly the presiding psychiatrists Southard and Solomon - to promote psychiatry as a means of scrutinising the everyday life of patients. The model of mental health care which this hospital - the first of its kind - promoted was important, representing as it did an institution 'mid-way between asylum practice and private practice ... extending its benefits to a group of sick persons far removed from the medicolegal concept of 'insanity''[104] In this context, Lunbeck's chief interest in neurosyphilis concerns the possibilities that it offered in opening up the everyday personal lives of patients to scrutiny - giving psychiatrists 'license to bring sex into psychiatry'.[105] Although she also notes the importance of the medical disease paradigm to which neurosyphilis conformed, she does not draw out how closely the project of normalisation and the tools of laboratory medicine were allied.

In 1917, Southard and Solomon published their comprehensive guide to the diagnosis and management of neurosyphilis, based upon their work at the hospital. In the preface appeared their optimistic statement: 'Syphilis is in a sense the making of psychiatry and will go far to pushing psychiatry into general practice'.[106] Although they were not explicit as to how syphilis was the making of their speciality, the book itself helped to explain, since it was in many ways a dedication to the Wassermann test. In each of the case histories, the test was used to establish the diagnosis in the

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[102] See K Jones, Asylums and After (London and Atlantic Highlands, N J: The Athlone Press, 1993), pp 126 - 140.
[103] Lunbeck, 1994.
[104] Southard and Solomon, 1917, p 430.
[105] Lunbeck, 1994, p 54.

face of obscure or vague symptoms. Often such cases were termed 'paresis sine paresi': incipient disease in which patients had absolutely no clinical signs or symptoms of GPI, but yielded positive laboratory results. The book described how the test allowed the hospital's psychiatrists and social workers to extend their practice to those in the surrounding community. With its aid they could survey, monitor, and advise those who would not formerly have been considered patients, since they were not suitable for certification. They could, for example, assess the families and associates of patients with neurosyphilis; thus the test might reveal the seeds of insanity in the 'normal-looking family' of a known syphilitic - whether in an unknowingly infected wife or in a child with latent or tardive hereditary syphilis. They could also closely monitor when a neurosyphilitic patient might be considered safe to marry. The test was thus central to the programme of extending psychiatry into the social sphere of the everyday: 'The work in psychopathic hospitals upon neurosyphilis ... is essentially a part of the public health programme, although our special work will not soon be taken over by the public health officers, so complicated are the ramifications of medical and social diagnosis and treatment in the neurosyphilis group ... The psychopathic hospital and asylum out-patient departments tap another reservoir of syphilitic families at a stage when the initial horror of syphilitic infection is dimmed or erased.'[107]

In Britain, similar efforts during the same period concentrated upon the detection and management of early disease. George Robertson expressed the underlying fear eloquently: 'General paralysis is common in our large cities, and

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[106] Southard and Solomon, 1917, p 8 (preface).

assumes so many disguises that it is necessary to be ever on the alert for it ... A man whose sanity is not yet questioned scandalises his neighbours and ruins his good name by his conduct in public places, or he dissipates his means and brings his family to want by senseless extravagance or by muddling his affairs. Nothing more need be said to indicate the value of an early diagnosis of this disease ...' Testing the cerebro-spinal fluid was, he suggested, the only objective way of identifying such people who - although not overtly insane - had the seeds of insanity within them: 'Are [such tests] not the most important element, and would we not be justified in diagnosing the disease from their presence alone? They are present at a very early stage, exactly how early no-one yet knows, and it is quite possible these reactions and signs may exist before there are noticeable clinical symptoms ... If ... a patient's blood and cerebro-spinal fluid gave a double positive Wassermann reaction, associated with lymphocytosis, plasma-cells, albumen, and an increase of globulin, it would scarcely be possible, in my opinion, to avoid the diagnosis of general paralysis ... even in the absence of any definite psychological or neurological symptom.'[108]

Scrutiny of the cerebro-spinal fluid was, in fact, pushing the limits of neurosyphilis beyond even the point at which the Wassermann test was positive. A large number of studies during the early twentieth century suggested that syphilis might attack the central nervous system in the very first weeks of infection. As early as 1903, the French pathologist Ravaut reported that, of 100 cases of early secondary syphilis, 72 showed pathological cellular changes in the CSF. This research was not taken up in Britain until around 1911, when scientists became interested in the effects

[107] Southard and Solomon, 1917, p 431.

of the new arsenical treatment '606'. From this period onwards a large number of investigations revealed various CSF changes (increased pressure, cell excess, rise in globulin content), with or without positive Wassermann tests, in newly diagnosed syphilitics. Surgeon-Lieutenant Fildes, exhaustively summarising the research to date in 1920, commented: 'Looking at these findings as a whole, it is certain that a very considerable proportion of cases of early syphilis have obviously abnormal cerebrospinal fluids and a very much higher proportion possibly abnormal CSFs ... whilst many show no discoverable [clinical] signs of disease at all'.[109]

The precise meaning of such studies remained under debate. It seemed unlikely that the CSF changes necessarily denoted neurosyphilis, since a smaller proportion of syphilitics went on to develop overt neurosyphilis than revealed the changes. However, firm evidence was not available, since no follow-up studies of such patients were carried out during the 1920's or 1930's to ascertain their outcome; a situation which frustrated many in the psychiatric profession. The important point taken by most doctors was that the changes denoted the threat of later GPI or tabes. These more subtle changes in the CSF, curiously, rendered even the Wassermann test too blunt a tool to indicate the presence of danger: 'While one waits for the blood Wassermann to become definitely positive ... the sprirochaetes are intrenching themselves in the nervous and other systems ... If the number of organisms reaching the meninges is small, or if the cells of the host fail to react, it will naturally follow

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[108] Robertson, 1913, p 204. My italics.
[109] P Fildes, R J Parnell and H B Maitland, 'MRC Special Report Series, No 45: Unsuspected Involvement of the Central Nervous System in Syphilis', 1920. PRO, Kew: FD4/45, pp 10 - 11.

that there will be no sign of nervous infection and the spinal fluid will be normal, although spirochaetes may be locked away in the fastnesses of the nervous system'[110]

In theory, pushing back the starting-point of neurosyphilis offered great opportunities for non-asylum surveillance and management. Calls for the early diagnosis and vigorous treatment of syphilis itself took on an added urgency; since as well as rendering a patient non-infective, early intervention - it was hoped - might prevent a subsequent attack on the nervous system.[111] The scourge of hidden syphilis was of particular relevance, since it was assumed that most of those who developed neurosyphilis had suffered from subclinical rather than overt syphilis - including disease which had been inadequately treated and wrongly believed to be cured. There were increasingly recommendations that every patient who tested positive for syphilis should also have regular lumbar punctures to detect incipient nervous spread. One commentator enthusiastically recommended two-yearly lumbar punctures over ten to fifteen years - and the energetic treatment of positive results with 'every means at our disposal'.[112] The greatest vigilance was to be placed upon the bearers of such results - often regarded as time-bombs which might erupt into overt disease at any time: 'It is necessary for these persons to lead very quiet lives, avoiding all sources of nervous or mental excitement, or exhaustion and the use of alcohol.'[113] Until the 1920's, the intention was to vigorously treat such positive test results in the hope of reversing the

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[110] R Ironside, 'The Prophylaxis of Neuro-Syphilis', Brit J Ven Dis, Oct 1927, Vol 3, No 4, pp 273 - 289; Discussion pp 290 - 298; on p 275.

[111] As Robertson commented hopefully: 'Sufficient time has not elapsed to enable anyone to say that a complete cure of syphilis by salvarsan, with a permanently negative Wassermann reaction, will prevent the development of general paralysis, but it is reasonable to think so ...': Robertson, 1913, p 213; see also T B Hyslop, 'General Paralysis', in J W Ballantyne (ed), Encyclopaedia Medica, 2e, 10 Vols (Edinburgh and London: W Green and Son, 1917), Vol 5, pp 286-96; on p 296.

[112] Ironside, 'Prophylaxis of Neurosyphilis', 1927, p 277.

reaction, for once the degenerative process of GPI had taken hold, the outlook was bleak. In contrast to claims for early syphilis, treatment of GPI by salvarsan was admitted to be useless. Robertson's recommended treatment during the period - intra-venous and intra-spinal salvarsan, intra-spinal anti-syphilitic serum, urotropine and calomel - produced, he confessed, negligible results; in none of his patients did the Wassermann reaction become negative for any length of time, and this disappointing finding was echoed by practically all of those who reported results.[114]

British psychiatrists, however, did not visibly succeed in using the test to the same extent as their American colleagues in Boston. Theoretically private practice was the area most amenable to surveillance of patients. Here, closer doctor-patient relationships over periods of time, easier access to financial resources, and greater guarantees of privacy, meant that both parties had incentives to co-operate with recommended protocols. Reality, however, is extremely difficult to assess, since most doctors wrote about what they thought it desirable to do, rather than what they achieved in practice. However, some doctors were clearly concerned about their own inadequate diagnosis of syphilis and neurosyphilis. In 1924 one practitioner typically appealed for his colleagues to be more astute in making the early laboratory diagnosis of GPI since, he assumed, it would fall to them to pick up these early cases amongst the apparently normal population. All practitioners, he commented, should learn to perform the lumbar puncture in order to carry out this vital task.[115]

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[113] Robertson, 1913, p 214.
[114] Robertson, 1913, pp 216 - 219; see also Mott, 1915, p 177.
[115] G W T H Fleming, 'The early diagnosis of General Paralysis', Practitioner, 1924, 112, pp 287 - 295. See the similar appeal by an anonymous practitioner for his colleagues to make a fuller use of the Wassermann test in order to pick up the 'increasingly unusual or hidden manifestations of syphilis'

In public practice, it is easier to appreciate the obstacles to surveillance of early neurosyphilitics. Most obviously, any surveillance that did take place fell more naturally to venereologists rather than psychiatrists - and here, problems of organising regular follow-up seemed insurmountable. During the War, the Navy was one of the few sites in which a large number of men with primary syphilis had lumbar punctures according to recommendations; ten years later, however, the fate of such patients over the long term was unknown.[116] Even after the introduction of the VD clinics, attending patients were notoriously reluctant to submit to follow-up checks; and it was naturally difficult to persuade them to undergo a painful lumbar puncture for a second time - let alone repeatedly over years! Doctors accepted that patients who felt healthy were loathe to undergo such intervention, and that the 'human element' of treatment often meant that ideals had to be sacrificed. A St Paul's venereologist wrote in 1927 that in up-to-date clinics it was imperative to test the CSF in all new syphilitics. He conceded, however: 'The performance of a lumbar puncture following upon scraping of the chancre, possible gland puncture, taking of the blood specimen for the Wassermann test, intravenous injection of arsenobenzol, deep subcutaneous or intramuscular injection of bismuth or mercury plus massage, might be calculated to increase the incidence of treatment defaulters ...'[117] 'The clinician', commented another doctor, 'should beware of adding to [fear of syphilis] by injudicious and unnecessary repetition of lumbar puncture and over-treatment ... "A syphilitic patient

found in the community: Anon, 'The Wassermann test in General Practice'. Lancet, Mar 18, 1922, p 546.

[116] R N Ironside, 'Meeting of the Medical Society for the Study of Venereal Diseases', Lancet, May 14, 1927, pp 1025-6.

should not be turned into a laboratory animal"'.[118] For these reasons - as well as caution about the safety of lumbar puncture - there was never a clear consensus amongst doctors as to even the desirability of routine invasive follow-up.

At the National Hospital Queen Square early cases of tabes dorsalis and tabo-paralysis, and sometimes recent-onset cases of GPI, were admitted and treated vigorously. In 1922 the typical protocol was as follows: '[The patient's] cerebro-spinal fluid and blood are taken for examination. He then starts on a course of iodide, mercury, and ... intravenous novarsenobenzol. For occasional cases we give intrathecal injections ... of mercurialized serum ... With this we combine re-education, massage, rest, and such symptomatic treatment as special cases may require.'[119] Laboratory studies showed, however, that at the point of admission the disease was practically always too far advanced to reverse clinical features of test results.

For most hard-pressed asylums there was far less leeway for admitting and treating early - let alone subclinical - disease. Even at the most favourable site - the Maudsley Hospital - general comments suggest that the model of non-asylum care did not always work in practice. In 1924 the superintendent Mapother complained about the increasing pressure on beds, which meant that admissions were frequently unsuitable: patients in quieter wards were not 'mental' at all, and those in acute

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[117] C H Mills, 'Routine Examination of the Cerebro-Spinal Fluid in Syphilis', BMJ, Sept 24, 1927, pp 527 - 532.

[118] G H Monrad-Krohn, 'Some Settled and Unsettled Problems in Neurosyphilis', in J R Lord (ed), Contributions to Psychiatry, Neurology and Sociology: Dedicated to the Late Sir Frederick Mott (London: H K Lewis and Co Ltd, 1929), pp 295 - 301; on p 296. See also Report of Meeting of the BMA Section of Venereal Diseases: 'Routine Examination of Cerebro-Spinal Fluid in Syphilis', Lancet, Sept 10, 1927, pp 553-4: 'It was essential' commented the member of one meeting on this subject, 'not to lose sight of the patient himself when dealing with his blood and cerebro-spinal fluid'.

[119] E M Dunlop, 'The Wassermann Reaction in the Diagnosis and Control of Treatment of Neuro-syphilis'; and discussion, BMJ, Oct 7, 1922, pp 632-5; on p 635.

wards were 'no different from [in] an ordinary mental hospital'.[120] More specifically, it is difficult to judge how extensively the Wassermann test was used here for either diagnosis or monitoring of treatment. Figures are available for the numbers of routine tests performed at the attached LCC laboratory (during 1931, for example, 5254 sera and 1328 CSF samples were tested); however, these figures represent routine work for the whole of the London area served by the laboratory. Annual reports show that the majority of patients treated in the out-patient department were diagnosed as suffering from dementia praecox, anxiety, depression, or neurasthenia.[121] Treatments offered were limited, but increasingly consisted of various forms of psychotherapy. On average, less than 1% of cases were diagnosed as general paralytics and managed solely as out-patients; and it is possible that these were early neurosyphilitics who were treated according to recommendations.[122] Details of follow-up, however, are not available - although staff continued to feel disappointed by the difficulties of following up discharged patients in general. More commonly, general paralytics were admitted from the out-patient department to the wards, or straight to the wards for management as in-patients. They still made up a very small proportion of total admissions; and although it is possible that they included early cases admitted for anti-syphilitic treatment, a number were severe cases which were admitted simply for their teaching value.

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[120] Annual Report of the Maudsley Hospital for the year ended 31 Jan, 1925, p 1. Bethlem Royal and Maudsley archives.

[121] During 1923, for example, the percentages for 560 out-patients were as follows: dementia praecox - 15%; anxiety states - 14%; depression - 14%; neurasthenia - 7%; amentia - 7%; paranoia - 7%; epilepsy - 6%: Annual report of the Maudsley Hospital for the year ended 31 Jan 1924.

[122] During 1923, 4 out of 560 out-patients were diagnosed with GPI.

We can conclude, then, that there was a considerable gap between the symbolism of the Wassermann test as a tool of psychiatry expansion, and its practical application. After the passing of the 1930 Mental Health Act, increasing numbers of psychiatric out-patient clinics opened. Although the Maudsley was therefore no longer the only such facility, it was still unique in being geared specifically to voluntary patients - especially since pressure of beds prevented many ordinary mental hospitals from admitting large numbers of voluntary patients. During this period, too, a more promising treatment for early neurosyphilis arrived. Malaria therapy, introduced during the 1920's, would intensify efforts at surveillance of early disease, and would become far more closely bound to hopes of moving psychiatry away from the asylum and into the community. In this area practice approached ideals more closely, and malaria therapy became a more potent indicator of the possibilities of non-asylum psychiatry than had the Wassermann test alone.

Stretching the Boundaries of GPI

Psychiatrists, I have suggested, expressed far less antagonism towards the Wassermann test after its introduction than did their general medical and venereology colleagues. The value of laboratory rhetoric to a frequently insecure specialty is one possible explanation for this; although it is equally possible that they had fewer concrete management decisions to make on its basis, and so were less hindered by its technical difficulties. Some retrospective criticisms, however, did surface towards the end of the 1920's. One such was published in 1930 by Power, the superintendent of Brent Wood asylum. His concern was that the test had caused such an obsession with

the syphilitic cause of GPI that interest in the family and alcoholic histories of asylum patients had waned, stifling an open-minded spirit of enquiry.[123] Power was rather a lone voice, and the article did not contribute towards a major debate.

A more important discussion, however, occurred in 1928 during the course of a long meeting of the Medico-Psychological Association; and it provided an interesting contrast to earlier claims that the test had tightened the diagnosis of the disease. Psychiatrists agreed that the clinical features of GPI appeared to have become increasingly more diverse - particularly since the War. Although the consensus was that this reflected a real change in the manifestation of the disorder, some felt that the test itself was implicated in the trend. The psychiatrist John Brander (superintendent of Colney Hatch Asylum) was a fervent exponent of the view that the identity of GPI - confidently established by the clinical methods of the older generation of alienists - was now being broadened and muddied because of an over-zealous faith in the laboratory. 'Let it be remembered', he pointed out, 'that the diagnostic criteria of this disease and its fatal progression were developed many years before the days of the ... Wassermann test ... In recent years any mental disease - whatever the history, whatever the physical signs, if it didn't respond to treatment and showed a persistent Wassermann reaction - was general paralysis ... the diagnosis has been made often on the strength of a single blood examination - with less regard for clinical features.'[124]

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[123] Power, 1930, p 525.

[124] J Brander, 'The Diagnosis of GPI as a Clinical and Pathological Entity', J Ment Sci, 1928, 74, pp 673 - 682; on p 675.

Brander's objections reflected a suspicion of over-valuing the laboratory; but they also demonstrated that, to some at least, the question of how GPI should be defined had not been closed by the advent of microbiological and laboratory criteria. They were extended to an interesting echo of arguments used against the syphilitic origin of GPI during the nineteenth century. Brander suggested that cures (through malaria therapy) were being wrongly claimed for these new Wassermann-defined forms of GPI - leading to an unjustified therapeutic optimism. GPI was, he insisted, by definition an incurable disease, and when diagnosed on clinical grounds alone was fatal in practically 100% of cases. The large number of current cases which improved, reacted to malaria therapy, or remained stationary for long periods of time were simply not GPI.

The General Paralysis Sub-Committee of the MPA took up Brander's charges in the following year, but did not uphold them.[125] Practically all of the psychiatrists involved protested that they took a cautious attitude towards laboratory methods, and acknowledged the Wassermann test only as an adjunct, or confirmation, of clinical findings. The President summarised: 'Surely laboratory technique must always have a subordinate place in diagnosis, and could never supplant the results obtained from careful clinical examination'.[126] The mistakes that Brander referred to, the meeting decided, had occurred back in 1910 - 12 when the test was new, and 'some modern men had placed too much stress on laboratory findings.'[127] Since then things had changed, and 'the present teaching was for the clinical findings to have the

[125] See 'General Paralysis: A Discussion ...', J Ment Sci, 1929.
[126] Ibid, p 11.
[127] Ibid, p 290.

predominance every time'[128] It was generally agreed that more varied clinical phenomena were now being treated as GPI; but whereas Brander maintained that this was due to changes in diagnosis directly resulting from over-reliance on the Wassermann reaction, the majority contended that the disease was indeed manifesting in new forms, many of which were treatable: 'The term 'general paralysis' has ... gradually come to include numerous cases with atypical symptomatology and course.'[129] Perhaps, suggested Lord, the views of Brander and his colleagues were simply designed to belittle the encouraging results of malaria therapy: '... One could always argue like that of any advance in medicine. Let them take care not to repeat London's reception of the Lister antiseptic treatment of surgical cases, and at some future time have to look back upon the delaying of good work for humanity with regret and not a little shame.'[130] Nevertheless, the General Paralysis Sub-Committee resolved to establish a national register of general paralytics, with their symptoms, test results, and response to treatment, in order to clarify how the disease should be classified.

Brander certainly seems to have represented the minority view in this particular discussion; nevertheless other psychiatrists were prepared to support him in the following years. The superintendent of Horton Mental Hospital commented in 1935: 'It was first thought that diagnosis would be made easier, but on the contrary, reliance on the serological findings has led to confusion and wrong diagnosis, with

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[128] Ibid, p 280.
[129] Ibid, p 22. I return to this theme in Chapter 5 below.
[130] Ibid, p 22.

consequent fallacious statistical results.'[131] The majority of psychiatrists, nonetheless, claimed to be well aware of the dangers of over-reliance upon laboratory medicine, and eager to deny that they were guilty. Again, it is difficult to substantiate the claims of Brander and his supporters. Patterns of diagnosis from national to asylum levels, as we have seen, show no discontinuity which might suggest that there was a major change in the numbers of patients diagnosed as a result of the Wassermann test. In particular, from 1912 there was a steady fall in both deaths and asylum admissions attributed to GPI which does not support a wholesale broadening of the diagnosis. Granted, the critics of the test might well have referred primarily to diagnostic practice in sites apart from the asylum. This became increasingly important after the 1920's; but practice is well-nigh impossible to assess due to lack of data. Historical caution would probably suggest that over- and under-diagnoses due to the test (compared to pre-test diagnoses) cancelled each other out, so that there was no overall change in the trend of figures for GPI: thus the undoubted perceptual changes that laboratory medicine brought were not sufficient to render figures incommensurable before and after the Wassermann era.

On an individual level, psychiatrists were understandably often ambivalent as to how they should incorporate the new laboratory tools into their practice. We certainly cannot take at face value claims - whether contemporary or retrospective - that the test revolutionised the management of GPI and was fully accepted by practitioners; nor that it was seized indiscriminately simply because it represented the excitement and status of laboratory medicine. Overall I would suggest that the real

[131] W D Nicol and E L Hutton, 'Some Clinical Aspects of General Paralysis', J Ment Sci, 1935, 81, pp

importance of the test lay in its symbolic rather than its practical power. First, it persuaded psychiatrists of the syphilitic aetiology of GPI in a far more concrete way than had Fournier's statistics. Second, it undoubtedly became part of the 'culture' of psychiatry, and as such must have had a huge effect upon individual patients' experience of their disease; in particular it turned doctors' attention to the possibilities of scrutinising subclinical illness - despite the fact that there was clearly a considerable gap between the ideals of surveillance and early treatment, and practical delivery. Third, it was used as a powerful symbol of the threat of syphilis and - despite some contemporaries' criticisms - of the triumph of scientific advance.

But this latter point threw up the contradictions inherent in the test particularly clearly. Even as psychiatrists held up the Wassermann reaction as a symbol of their hopes for scientific psychiatry, they seemed to express little confidence that the fortunes of their specialty were changing in the years after its introduction. In 1909 Mercier, the President of the Medico-Psychological Association, sent a letter to all of the medical school examining boards, condemning the poor position of psychiatry with respect to medicine, and stressing the urgent need for organised post-graduate teaching. The aim, as always: 'To place the teaching of psychiatry on a solid, scientific basis, and so bring it into line with other special departments of medicine in this country.'[132] By the mid-1910's the response was reported to be poor, and the same complaints arose time and again: that psychiatry in Britain was undervalued and isolated in comparison with the continent; that it was wasting the opportunity for

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804 - 821; on p 805.

[132] Quoted in A Lewis, The State of Psychiatry: Essays and Addresses (London: Routledge and Kegan Paul, 1967), p 117.

scientific research; that it must re-mould itself as a branch of public health and reorganise itself institutionally to separate acute from custodial care.[133] An article published in the Lancet in 1913 expressed no excitement that scientific medicine was offering new hope to British psychiatry; rather it called upon the speciality to awake from its lethargy: 'British psychiatry can no longer revolve in an eddying backwater whilst the current of scientific research is flowing strongly past it.'[134] In 1920, a further encyclical to the universities reiterated the complaint that psychiatry had seen insufficient improvement in its status and had not, as hoped, been brought into line with mainstream medicine. Rhetoric in journals can, of course, only tell us a small part about how the Wassermann test in particular, and laboratory medicine in general, was perceived to be commensurate with the development of psychiatry. Nevertheless, observations such as these might cause us to look more carefully at the assumption that such a tool was necessarily successful in aiding such developments. Ambivalence about the progress of the specialty, an uneasy mixture of hope and frustration, was simply the continuation of a long and familiar tradition in psychiatry.

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[133] See, for example, Leader, Lancet , Apr 6, 1912, pp 934-5, and the series of letters which followed, including Bedford Pierce, Lancet, Apr 13, 1912, pp 1017-18; G Gibson, Lancet, June 2, 1912, p 1717-18.

[134] Anon Leader: 'Research work in Insanity and the Study of Curative Treatment of the Insane', Lancet, Nov 8, 1913, p 1333.

CHAPTER 4

'DESPERATE ILLS DEMAND DESPERATE REMEDIES':

MALARIA THERAPY in BRITAIN 1920 to 1950

Introduction

During the first two decades of the twentieth century, the identity of GPI as an almost invariably fatal disease remained unchallenged. Once the patient had entered an asylum, good nursing care was agreed to be the only real help that could be offered; and although anti-syphilitic treatments such as mercury and potassium iodide were sometimes recommended, few claimed any lasting benefit from their use. In 1910, Erlich's anti-syphilitic arsenical compound Salvarsan ('606') was introduced to great excitement; an excitement increasingly tempered by disappointment and even hostility over the following years. Both Salvarsan and its successor Neosalvarsan were used to treat general paralytics through a variety of intravenous and intraspinal techniques; but here too they failed to live up to their initial promise: 'It seems', wrote Mott in 1915, 'that GPI hasn't been cured, or even much benefited, by salvarsan and neo-salvarsan, intravenous or intrathecal ...'[1]

During the early 1920's, however, a new treatment was introduced from Europe which seemed a world apart from Ehrlich's painstaking experimental work. Malaria therapy had been developed initially as a result of chance observations, and its theoretical basis was a mystery. Nevertheless it appeared to have a dramatic effect

upon the course of GPI, and from 1922 it was rapidly implemented by the more enthusiastic superintendents of British asylums.

There have been few comprehensive historical accounts of malaria therapy; and fewer still in the British context. Those that we do have are written predominantly by doctors, and generally fall into one of two categories. First there are a number of straightforward, non-analytical histories of the therapy, usually triumphalist in tone: Purdon-Martin, for example, entitles his short history: 'The Conquest of General Paralysis'; Rollin, who offers useful information about the beginning of malaria therapy in Britain, is nonetheless mainly concerned with eulogising the Horton Malaria Laboratory.[2] Whitrow's biography of Wagner-Jauregg considers in detail the Austrian psychiatrist's development of malaria therapy - and in particular his years of experimentation leading up to the 1920's. Again, however, her prime concern is to eulogise her subject: there is disappointingly little consideration of the wider reception and application of his work; and only passing reference to the ethical questions which affected his career at least once - in 1924 - when the Nobel Prize was refused him because one of the judges considered his treatment tantamount to a crime.[3]

Revisionist historians, too, have been strangely silent upon this episode; nonetheless it has commonly been seen as the herald of physical treatment in psychiatry, and as such has sometimes been cast as a coercive, ethically and scientifically unsound practice. Austin et al., for example, comment that: 'Ethical

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[1] Mott, 1915, p 179.
[2] Purdon Martin, 1972; H R Rollin, 'The Horton Malaria Laboratory, Epsom, Surrey (1925 - 1975)', Journal of Medical Biography, 1994, 2, pp 94 - 97.

considerations did not constrain researchers from injecting known pathogens into mental patients...'[4] In particular they target the lack of scientific rigour with which the treatment was assessed - actually the failure of contemporary doctors to use the randomised controlled trial twenty years before its development! They draw lessons from this retrospective 'failure' for AIDS researchers today, who might be tempted to bypass testing procedures in their eagerness to promote hopeful treatments. Similarly Fennell, in his history of psychiatric treatment and the law, describes it as 'the Board [of Control's] first, rather inauspicious, foray into actively promoting a particular therapy', and concentrates upon its role as a sanction for the use of heroic non-consensual physical treatments in which 'actual efficacy ... was secondary'.[5]

Both interpretations are perhaps most useful in showing how professionals use their past to illustrate current concerns; but both - by adhering to a particular ideological stance - also fail to give a coherent historical account. None attempts to trace in detail the introduction and popularisation of the therapy from the point of view of inter-war doctors - and in particular the scientific and ethical discussions which the treatment stimulated. Certainly there were strong strains of triumphalism in psychiatrists' rhetoric about the new treatment; and certainly it was institutionalised successfully in Britain - particularly through the development of the Horton Treatment Centre. Psychiatrists were quick to acknowledge the value of a treatment which could make general paralytics - traditionally perceived as the most wretched and worthless of the asylum population - at least more manageable, and at best

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[3] Whitrow, 1994.
[4] Austin et al, 1992, p 518.
[5] P Fennell, Treatment without Consent (Routledge: London and New York, 1996), p 127.

productive members of society again. Malaria offered them yet another opportunity of using GPI to make claims for their specialty, expressed in terms of prestige, practical asylum logistics, and - more strongly than ever before - in terms of benefits for individual patients.

But once again, paying attention to practice as well as rhetoric emphasises the complexity of doctors' attitudes towards the 'friendly fever'.[6] As we might easily predict, few regarded the therapy as wholly successful or unproblematic. Questions of efficacy, of assessment of results, and of possible dangers were constantly discussed - particularly as initial excitement wore off, and the long-term consequences of treatment were faced. Thus whilst the Board of Control spoke of malaria therapy as an 'established and proven treatment' in 1930, almost a half of British asylums were still not using it, for a variety of practical and ideological reasons. As questions of benefit, risk, and ethics were addressed, pragmatism - often expressed as a peculiarly British stance - emerged as the self-conscious rationale for the treatment; correspondingly rhetoric subtly changed from that of 'miracle cure' to that of a potentially hopeful advance. In unravelling the history of malaria therapy, it is once again doctors whose views are at the forefront. Nevertheless, malaria arrived during a period in which patients were increasingly targeted before and after their period of asylum confinement; consequently, for the first time it is possible to have more than a glimpse of what GPI and its management might have meant for the patient himself and for his family.

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[6] A term adopted in popular American parlance: see, for example, F Cross, 'Friendly Fever', Good Housekeeping, Feb 1935, 100, 2, pp 46-7.

The Background to Malaria therapy: Julius Wagner-Jauregg

Julius Wagner-Jauregg is traditionally the central figure of any discussion about malaria therapy. Although he himself acknowledged that the treatment was not his original observation, he nonetheless developed and popularised it throughout Europe, and was subsequently rewarded with the Nobel Prize for Medicine in 1928.[7] His observations upon the link between fever and psychosis began as early as 1880 at the Viennese Asylum of Lower Austria. Here, improvements in the symptoms of psychotic women following erisipelas infection sparked his interest in a subject which he would return to intermittently over the next forty years.[8] By the 1890's he was experimenting with a variety of deliberately induced fevers; but soon concentrated upon those caused by Koch's controversial tuberculin. His choice of a vaccine, he later wrote, was influenced by popular antagonism to human experimentation - since it allowed the production of fever without an accompanying infectious disease. Over the next twenty years he concentrated almost entirely upon tuberculin's effect on general paralysis: this condition, he felt, gave the most dramatic response; was one of the commonest insanities in Austria at the time; and was incurable, so that successful results were relatively easy to demonstrate. Accounts of his tuberculin trials - published intermittently before the First World War - claimed such results as 25% remission rates in treated patients, although few, he admitted, maintained their recovery. Neither did his work meet with much enthusiasm: his account at the 16th

[7] For a full account of Wagner-Jauregg's life, see Whitrow, 1994.

International Medical Congress of 1909 met with scepticism and only muted discussion. Wagner-Jauregg judged that malaria would produce far more successful results than tuberculin; for a long time, however, he remained wary of inducing 'live' infection in his patients, and continued to experiment instead with a variety of other vaccines. His account of the first malaria inoculation was dramatic. The 'psychological moment' which induced him to make the attempt occurred, he recounted, when a prominent oil engineer was admitted to the Vienna Psychiatric Hospital with general paralysis: 'It was the tragic outlook for this man which again forced on my mind the thought of producing intentionally an infectious disease ...'[9] At about the same time, in June of 1917, a soldier from the Macedonian front entered the hospital with symptoms of tertian malaria: 'This', Wagner-Jauregg recounted, 'I regarded as a sign of destiny because soldiers with malaria were usually not admitted to my wards ...'[10] He seized the opportunity, and inoculated nine GPI patients with the soldier's blood that summer - waiting a year 'to see whether this experiment would prove to be a real therapeutic success.'[11] Equivocal results did not deter him from repeating the experiment a year later, this time with blood obtained from a local military hospital. Whilst failure to examine the blood microscopically resulted in

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[8] His first article on the subject drew attention to many observations in past medical literature that fever could have a beneficial effect upon psychotic symptoms: J Wagner-Jauregg, 'Ueber die Einwirkung fieberhafter Erkrankungen auf Psychosen', Jahrb f Psychiat u Neurol, 7:94, 1887.

[9] J Wagner-Jauregg, 'The History of the Malaria Treatment of General Paralysis', 1935, reprinted in Am J Psych, 151: 6, June 1994 supplement, pp 231- 4; with comment and translation by WL Bruetsch; on p 233.

[10] *Ibid*, p 233.

[11] Ibid.

three patients dying of malignant malaria, the fourth 'survived after large doses of quinine and neosalvarsan ... His paralysis was cured completely and lastingly.'[12]

The unfortunate deaths, he claimed, took away his appetite for malaria experimentation until a year later, when he co-operated with a colleague working upon the natural history of malaria. From this time, he began uninterrupted large-scale experimental treatment with malaria therapy at the Vienna Psychiatric clinic, with apparently increasingly successful - and safer - results. 'The malaria strain of September 1919', he wrote in 1935, 'has been maintained up to the present day - more than 16 years - in continuous human passage. I do not know of any other strain in the world which has been used for so many years.'[13] News of Wagner-Jauregg's work would reach Britain largely through the publications of his assistant Gerstmann, an optimistic advertiser of the therapy who routinely claimed 33% remission rates. Over the following years Austrian psychiatrists would place strong emphasis, too, upon the treatment of early cases; and even asymptomatic patients with positive Wassermann tests were soon treated at syphilis clinics in Vienna - work which continued on a far greater scale than was achieved in Britain.

Wagner-Jauregg's work is not the main subject of this chapter; nonetheless accounts of his innovation - particularly his own accounts - introduce points which would soon be relevant to British discussions. His experimental trials were marked by a characteristic attention to method and numerical detail. The majority of his studies compared treated and untreated groups of patients, a technique which he

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[12] Ibid.
[13] Ibid.

called the 'simultanmethode': one of his earliest trials, for example, entailed injecting sixty GPI patients with tuberculin, and comparing their life duration and remission rate with sixty untreated patients.[14] Evidently strongly interested in the importance of experimental design, he was said to have been frustrated by his inability to organise larger comparative trials; working at a short-stay clinic he had access to only small numbers of patients, and could not persuade larger asylums to co-operate. Such comparative trials would form the basis of the major British retrospective trial in 1928; and although carried out well before the development of the controlled trial, they demonstrated an attempt at rigorous testing. Wagner-Jauregg also had a strong sense of the rationale behind his experimentation. His professional interests suggest that he was primarily a pragmatist at a time when mainstream Austrian psychiatry inclined to the theoretical. His early ambition had been to take up internal medicine, and much of his work, such as that on cretinism and electrotherapy, was aimed at finding successful practical treatments for mental disorder: indeed he was described by one colleague (perhaps over-generously!) as being: 'the first to introduce to psychiatry the thought processes of internal medicine ...'[15] Nonetheless, he constantly defended his fever work as rational rather than empirical or serendipitous: 'We cannot be reproached for using a procedure which is irrational. We have listened to nature; we have attempted to imitate the method by which nature itself produces cures.'[16] Characterising his own work in this way suited his personality well. Wagner-Jauregg was a careful, reserved man, not given to over-statement or

[14] Ibid, p 232.
[15] Quotation of Potzl in Whitrow, 1994, p 63.
[16] Quoted in Whitrow, 1994, p 157.

enthusiasm. Papers upon his subject were dry, measured affairs rather than triumphalist advertisements for the treatment, and he set up a diffident image of himself to oppose the heroic account given by Paul de Kruif in Men Against Death.[17] Against the terrible background of the First World War, he stated, 'a therapeutic experiment could stir me little, in particular since its success could not be foreseen ... How sceptical I was toward the early successes with the malarial treatment is shown by the fact that I waited a year until the publication of the first report.'[18]

Despite all this, malaria therapy was obviously of a very different nature to the specific treatments promoted by laboratory science. It is true that the earlier tuberculin trials had been placed firmly within the tradition of bacteriology; and to Wagner-Jauregg the treatment's mysterious mechanism would surely be solved by laboratory science. However, the reception of malaria therapy in the 1920's indicated that dramatic non-specific treatments were now acceptable to psychiatrists - an attitude which Wagner-Jauregg believed signalled a change from previous years. Tuberculin therapy combined with mercury, he claimed, had given better results before the First World War than any other treatment, and yet had not been accepted: 'The medical scientists of that period were hypnotised by the discovery of the syphilitic aetiology of general paralysis and could see the solution of this special problem only in a specific treatment.'[19] Similarly in Britain - whilst many psychiatrists held an image of themselves as cautious and empiricist - malaria therapy would quickly take hold, and would become accepted by the establishment with little real concern about its

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[17] P de Kruif, Men Against Death (New York: Harcourt, Brace & Co, 1932): Ch 9: 'Wagner-Jauregg, "The Friendly Fever"'.
[18] Wagner-Jauregg, 1935, p 233.

theoretical respectability. This general acceptance pertained despite the concerns which psychiatrists would increasingly express about the efficacy and safety of the treatment.

When he began his experiments during the 1880's, Wagner-Jauregg recounted, he discontinued for a time because 'medical science of that period looked with disfavour at experimentation on human beings'.[20] Subsequently he recalled that he had continually delayed the use of malaria infection to replace vaccination because of similar ethical attitudes. Such concerns, however, do not seem to have affected the institutionalisation of malaria therapy during the 1920's. Wagner-Jauregg noted only one clear example of antagonism: the Nobel Prize, for which he was nominated in 1924, was refused because one member of the committee objected to the award: '[Gadelius] couldn't recommend the award to a physician who injected malaria into a paralytic because he was in his eyes a criminal ...'[21] Nonetheless the unanimous decision to award the Prize in 1927 (when Gadelius was no longer on the Board) demonstrated that mainstream endorsement of the therapy was not dampened by ethical misgivings.[22] This is not to say that there were no opponents to the therapy: Wagner-Jauregg had at least one high-level professional dispute with doctors who opposed what he was doing; and only thorough research of the Austrian context could uncover how widespread such antagonisms were. At the end of his life, however, he

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[19] Ibid, p 232.
[20] Ibid.
[21] Quotation of Wagner-Jauregg in M Whitrow, 'Wagner-Jauregg and Fever Therapy', Med Hist, 1990, 34, pp 294 -310.
[22] The Nobel Prize was only the culmination of his professional recognition; he was also awarded the Erb medal of the Society of German Neurologists in 1926; and Honorary membership of the German Society for Psychiatry in 1927 for his malaria work.

was happy to claim malaria therapy as his most important and personally satisfying work - and as the work that had brought him most honours and awards from his colleagues. In Britain, too, occasional ethical misgivings would be expressed; but a robustly pragmatic assessment of results would prevent the majority from treating them as a serious obstacle to practice.

Malaria therapy comes to Britain

During 1922, a number of British asylum superintendents were intrigued enough by Austrian reports to attempt malaria therapy themselves. Accounts in the British literature had captured the optimism of the Continental experience, promising dramatic and lasting recoveries in up to a third of patients treated: 'Most cases can be helped', the Journal of Mental Science reported, 'To early cases we can, with Wagner-Jauregg, offer a complete cure'.[23] The psychiatrist McAlister inoculated his first patient at the Edinburgh Morningside Asylum in March, after a series of logistic hurdles: 'The delay in getting the experiment under weigh [sic] was due to the difficulty of getting an uncomplicated case of benign tertian malaria. We ransacked the whole of Edinburgh and even applied to the School of Tropical Medicine in London in a vain effort to get a suitable case ... We had, however, the good fortune to admit a young man suffering from Dementia Praecox from whose blood in the course of a paroxysm we were able to isolate the tertian organisms ...'[24] Grant, the Medical

[23] E W Scripture, 'The Treatment of General Paralysis by Malaria: The Use of Speech Inscriptions for Early Diagnosis', J Ment Sci, 1923, 69, pp 77 - 83; on p 82; For a British review of the Austrian results, see W Yorke, 'Malaria Treatment of General Paralysis of the Insane', Lancet, Feb 27, 1926, pp 427 - 431.

[24] Letter from McAlister to Board of Control, 11 June, 1923. PRO, Kew: MH 51/697.

Officer of Whittingham Asylum, displayed a similar enthusiasm, inoculating his first patient in July: 'Wagner-Jauregg's malaria treatment of general paralysis ... first attracted our attention in the early months of 1922, and through the kindness of Professor Stevens of the Liverpool School of Tropical Diseases, who inoculated the first cases for us, we were able to commence this treatment ...'[25] During the same year psychiatrists at the City of London Asylum also made trials, followed closely by doctors at six other London asylums, as well as Rainhill, Cardiff, and Winwick.

Detailed recommendations for carrying out the treatment were continually discussed; but a basic protocol was soon developed, which was described by the psychiatrist Nicol in 1929.[26] A sample of blood infected with tertian malaria was injected (subcutaneously, intramuscularly, or intravenously) into the general paralytic patient. After an incubation period of about two weeks, the patient developed typical recurring febrile attacks with delirium and rigors - during which time he was put on a quarter-hourly to four-hourly temperature chart, cold-sponged to prevent excessive temperature rises, and subjected to daily blood examinations. After between eight and twelve of these paroxysms, he was treated with quinine - usually combined with specific anti-syphilitic treatment - to arrest the disease. Following the treatment, he was 'generally exhausted and anaemic', and was gradually built up again with tonics - sometimes to receive further infections at intervals.[27] The process was exhausting

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[25] A R Grant, 'The Treatment of General Paralysis by Malaria', BMJ, Oct 20, 1923, pp 698 - 700; on p 698.
[26] W D Nicol, 'The Treatment of General Paralysis by Malaria', Brit J Ven Dis, 1929, 5, 2, pp 85 - 101.
[27] Ibid, p 91.

and gruelling both for patients and for nurses, who were intensively involved with the treatment over lengthy periods of time.

Initial results of malaria therapy reflected to some extent the encouraging reports from the Continent. Many of the treated patients indeed appeared to improve - sometimes dramatically. Whittingham asylum, for example, published its preliminary results in 1923: of 40 patients treated in the past year, 3 had been discharged and able to take up their former employment; 3 previously 'wet and dirty in their habits' had improved markedly; 2 formerly confined to bed were now up and about. Practically every case was claimed to have benefited to some degree, and the authors were optimistic that an extended trial would be worth while.[28] Individual case histories accentuated the happy effects: 'Case IV, male aged 54: 'On admission the patient was simple and childish; was completely disoriented for time and place. He was in feeble physical condition. Since March of this year he had been confined to bed and he was unable to walk. He had marked tremors of his hands and was unable to do anything for himself. He was inoculated on June 5th. He continued to improve rapidly after inoculation and is now able to walk about. He still has tremors of the right hand, but he is able to work in the ward. His mental condition is much better; he is now oriented for time and place. The rapid amelioration of his mental and physical condition was commented on by his relatives.'[29]

These early trials were improvised, even haphazard, affairs, and the search for suitably infected blood called for both initiative and experimental enthusiasm. That

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[28] Grant, 1923.
[29] Ibid, p 700.

British doctors, often characterised as cautious conservatives, should have leapt into the experimental unknown was the source of some pride. The Board of Control commissioner Sir Hubert Bond later warmly described the Whittingham initiative: 'Should the high hopes so widely felt for the enduring success of this mode of treatment be confirmed, this brief statement as to how the treatment came to be started in England deserves a place here. It records a step which, having regard to our national susceptibilities, required no small courage to take.'[30] Early trials also marked the beginning of an alliance between psychiatrists and tropical disease specialists. The potential benefits to the latter were obvious: Warrington Yorke, Professor of Tropical Medicine at Liverpool, was closely involved in the first trials at Whittingham Asylum, and later recounted: 'When Dr Clarke [the deputy superintendent] ... first asked us to infect for him a number of cases of general paralysis, I had very little belief that it would do any good, but it at once occurred to me that if the alienists wished to call in the aid of a tropical disease to assist in the treatment of a mental disease, it was only right and proper that those whose interest lay in exotic diseases should seize the opportunity which the occasion presented, of making a number of much desired observations on malaria ...'[31] Both themes of empiricist boldness and co-operation between specialties would continue to characterise malaria therapy over the following decades.

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[30] H Bond, Preface to E T Meagher, General Paralysis and its Treatment by Induced Malaria (London: HMSO, 1929), p 24. PRO, Kew: MH 51/537.
[31] Yorke, 'Malaria Treatment ...', 1926, p 429.

Ministry, Board, and the Horton Treatment Centre

The Ministry of Health and the Board of Control became jointly involved in malaria therapy a year after individual doctors had made their first trials. The interests of the Ministry were primarily the implications of using and controlling malaria infection in Britain; those of the Board (successor to the Board of Commissioners in Lunacy) were primarily the implications for patient care and the advancement of psychiatry. Nonetheless, the concerns of both bodies overlapped, and they co-operated in much of their involvement with the treatment. Government intervention was spear-headed over the next twenty years not by a psychiatrist, but by the malariologist Colonel Sydney Price James. After a variety of posts in India, James had been appointed Medical Inspector and Adviser on Tropical Diseases to the Local Government Board (soon to become the Ministry of Health) during 1917. The advent of malaria therapy was fortuitous for him; much of his work over the next three decades sprang from the research opportunities that the treatment afforded, work centred at the Horton Malaria Laboratory which he established and headed until the mid-1930's. Despite fulfilling a large number of roles, it was for this research institution that James would be largely remembered - as his obituary by Christophers confirmed.[32]

Many of the steps taken by the Ministry and the Board after 1923 were instigated on the advice of James, who constantly expressed concerns about the potential hazards of the treatment, and urged the importance of close monitoring.

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[32] S R Christophers, 'Obituary of Sydney Price James 1870 - 1946', Obituary Notices of Fellows of the Royal Society, Mar 1947, Vol 5, pp 507-23.

During 1923 he was sent on a fact-finding mission to von Weber's clinic in Graz where the therapy was reported to be a success. He brought back impressions of a promising treatment which was nonetheless still very much in an experimental stage, requiring careful supervision: 'If it were desired to essay the treatment experimentally in England it could only be done in an Institution where the Ministry or other responsible Government Department could control the arrangements for the diagnosis of syphilis and for otherwise safeguarding the interests of the patients who act both as the subjects of treatment and as carriers of the particular strain of malaria parasites.'[33] The Board appeared to take his cautions seriously; within a few months the commissioner Bond had contacted all those asylums where he believed that trials were occurring, to enquire about their methods, complications of the treatment, precautions, and preliminary results. From this point, the Board tried to maintain contact with all the hospitals participating in trials, and kept a register of patients reported to have received malaria therapy.

Meanwhile, James' concern that malaria infection might spread outside asylum walls prompted him to make a series of experiments and hospital visits during the winter of 1923-4. His findings alerted him to another concern: despite evidently encouraging results, the number of patient deaths arising from malaria therapy seemed alarming, and James attributed them to the use of a particularly virulent strain of malaria. He prepared a series of guidelines which formed the basis for a circular sent by the Board to all asylum superintendents in 1924.[34] The circular warned that '...

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[33] Report by S P James copied to Board of Control file 25 May 1923. PRO, Kew: MH 51/697.
[34] O Dickinson (Secretary to the Board of Control of England and Wales), Circular letter to Medical Superintendents of County and Borough Mental Hospitals and Registered Hospitals in England and

already in some instances the induced malaria in the inoculated cases in England has been of an unusually severe type, accompanied by pronounced complications and with a tendency to fatality ...' [35] A number of precautions were suggested. Each asylum should be equipped with a laboratory suitable for the daily microscopic examination of the blood of inoculated patients. This daily examination - to be performed by a 'well-qualified' officer - would confirm that there were no malignant strains of parasite present in the blood, and would give warning of a rise in parasite numbers which might herald unsuspected complications. Patients should be monitored and nursed carefully during their illness by staff familiar with malaria, and should be thoroughly treated with quinine both during hospitalisation and after discharge, to prevent relapse. To prevent stray mosquitoes from biting patients and subsequently spreading malaria to the general public, all inoculated patients should be kept in mosquito-proof wards during treatment, monitored for several weeks after discharge, and notified to their local Medical Officer of Health. The Board promised to ensure that these procedures were being complied with, as part of regular inspection duties. It also offered short training courses in malaria therapy at the London School of Tropical Medicine; and twelve doctors participated in the first of these, held in June of 1924.

James' concerns also appeared to provide the impetus for establishing the Horton Treatment Centre - which would become the most important embodiment of Government interest in the therapy. James argued that a colony of mosquitoes,

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Wales, concerning the Malarial Treatment of General Paralysis (Feb 27, 1924), reprinted in J Ment Sci, 1924, 70, pp 337 - 341 (with enclosure of letter by P Mühlens on 'The Dangers of the Fever Treatment of Paralysis).

infected by a safe and controlled strain of malaria, might be bred at a central site of expertise: these mosquitoes could be used to inoculate patients at the centre, but also to supply asylums throughout the country. James explored a number of existing London asylums before settling upon the all-female Horton as the site of his new enterprise; and during the summer of 1924 the London County Council, Board of Control, and London Mental Hospitals Committee agreed that all women general paralytics from the London area should be concentrated here.[36] Part of the hospital was set aside as a mosquito-proof isolation unit, and the Ministry of Health provided the majority of funding for an on-site laboratory in which to establish the mosquito colony. Batches of female Anophiles mosquitoes were collected from the countryside around London (a search focusing particularly on pigsties), fed on malarial subjects, and incubated ready for breeding. Each patient could then be infected by placing the mosquitoes in a glass jar covered with netting, applying it to the thigh, and waiting for a bite.

The enterprise did not get off the ground easily. At the end of 1925, James was having great difficulty in starting and maintaining his malarial strain of P vivax - harvested from a patient who had contracted the disease in India - since London hospitals were failing to transfer a sufficient number of patients. The Board had several suggestions, such as inoculating dementia praecox patients to increase the critical mass of infection, or opening a male ward.[37] However, patients from around London were gradually transferred and treated in increasing numbers: 16 in 1925; 23

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[35] Ibid p 338.
[36] See Reports of the LCC, 29 July and 12 Dec 1924. GLRO: LCC/MIN 1146; also Letter from LCC to Board of Control, 1 Aug 1924. PRO, Kew: MH 51/698.

in 1926; and 39 in 1927.[38] James' subsequent 'Madagascar' strain, started during 1925, was passed successfully from mosquitoes to patients for many years. He took on a laboratory assistant, Percy George Shute, and between them they prepared inoculations for asylums all over Britain, making numerous visits to deliver mosquitoes in glass containers and to supervise treatment. A second Board circular of 1926 advised asylum superintendents that inoculation by mosquito was safer and more effective than blood inoculation, and advertised the Horton facilities for supplying both mosquitoes and education.[39] Many asylums, however, continued to use the less distasteful blood inoculation, which they achieved by co-operating with local tropical disease centres and by setting up their own chains of patient-to-patient infection.

Production of inoculations remained the primary function of the Horton laboratory, and the accompanying treatment hospital was quickly recognised as a centre of excellence for malaria therapy where medical and nursing techniques could be perfected to produce the best results in the country. Besides this, though, the Horton increasingly yielded research into malaria itself; thus also gaining a reputation as Britain's leading research centre for malariology. This aspect I shall return to when I consider the relationship between malaria therapy and tropical medicine later in the chapter.

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[37] Minutes of Board of Control meeting, Oct, 1925. PRO, Kew: MH 51/698.
[38] These figures are taken from the Horton clinical records: Clinical case-books of Horton Hospital (1925 to 1940). Archives of the Royal College of Physicians. There is a discrepancy in that James reported higher figures, but these seem to include treatment requests from other asylums. See SP James, assisted by P G Shute, Report on the first results of laboratory work on malaria in England (League of Nations, Geneva, 1926). PRO, Kew: CH/Malaria/57 (I).
[39] Malarial Treatment Circular 680, 8 Feb 1926. PRO, Kew: MH 51/240.

The Benefits of Malaria

Well before the establishment of the Horton Centre, London and provincial asylums had taken up malaria therapy in increasing numbers, and had reported encouraging - although highly variable - results. Of eight early studies published between 1923 and 1925, 'improvement' was claimed in proportions ranging from 14 - 62% of treated patients; of these, a proportion ranging from 8 - 28% were reported fit for discharge.[40] As further results were gathered, the figures became more consistent. A retrospective survey of all London trials, published by the LCC in 1926, claimed that an average of 33.5% of treated patients had been discharged, compared with only 2.6% of untreated patients. Furthermore: '... All the Medical Superintendents are agreed that in the majority of cases treated ... which proved to be unfit for discharge, a marked improvement in general well-being, cleanliness, and conduct was observed.'[41]

A strain of triumphalism certainly ran through these early accounts, as doctors hailed the first psychiatric treatment which appeared to have a tangible effect. Malaria therapy gave instant, practical results: patients, after all, who would normally rapidly die were being discharged from the care of the asylum. Enthusiastic case-reports provided testimonies to the exciting results; Lord, superintendent of the Horton, maintained that 'his experience in this matter was one of the most remarkable

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[40] These early reports were from Whittingham, Winwick, Morningside, the National Hospital Queen Square, Hanwell, Claybury, Belfast, and Dublin. For a review of trials at British hospitals up to 1926 see Anon, 'Research in Mental Disease', Lancet, Dec 25, 1926, pp 1338-9.

[41] 'The Value of Malaria Therapy in Dementia Paralytica: Preliminary Report from the London County Mental Hospitals Service', BMJ, Oct 2, 1926, p 603. See also Austin et al., who provide comparative results from 17 sources in Europe and the US: Austin et al., 1992, p 518.

and amazing he had ever seen'[42] The venereologist and pathologist Wansey Bayly described a woman admitted to the Horton at death's door: '... demented, unable to feed herself, emaciated ... and incontinent ... Imagine my astonishment when early this month as I was walking along Marylebone Road this lady, whom I imagined had been dead for some months, came up and addressed me, looking well, speaking quite sensibly, and walking with a firm and healthy gait. She informed me that malaria therapy had cured her ... I expressed astonishment at her miraculous recovery ...'[43]

How malaria might produce such dramatic effects remained open to debate. During the 1920's there were three main schools of thought: that it acted non-specifically through the pyrexia that it produced; that it enabled the body to overcome the activities of the spirochete by generally stimulating the metabolism and immunological defences; or that it acted through direct antagonism between the malaria parasite and the spirochete. The use of such a theoretically hazy procedure was accepted readily - and indeed was often portrayed as heroic: 'By a daring experiment in clinical medicine the treatment of the disorder has been advanced beyond all hopes, but the empirical method here has owed nothing to the discovery of the spirochaetal origin of the disorder ...'[44] The psychiatrist Graham echoed this cheerfully pragmatic acceptance: 'He had no doubt that this was a marvellous treatment, although it might not appear very scientific'[45]

But the problem of <u>defining</u> outcomes was a constant stumbling-block for psychiatrists. The terms 'cure' and 'remission' were often used interchangeably to

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[42] 'General Paralysis: A Discussion ... ', <u>J Ment Sci</u>, 1929, p 22.
[43] G Riddoch, 'Neurosyphilis', <u>Brit J Ven Dis</u>, 1928, 4, 1, pp 1 - 24; on pp 19 - 20.
[44] Leader, 'Early Treatment in GPI', <u>Lancet</u>, Jan 5, 1935, pp 34-5.

describe the best results of the therapy; yet few were prepared to assert that malaria was indeed strictly curing general paralysis. All but the earliest cases were assumed to have structural brain changes which could not be reversed by malaria, notwithstanding the sometimes vivid clinical evidence. The Wassermann test was a potential criterion for deciding whether cases were indeed being cured; however, although 'improvements' in the CSF profile of treated patients were reported (for example, reduction in lymphocyte and globulin counts), no firm correlation between clinical and serological results were confirmed. By the 1920's, in addition, doubts were being raised about the reliability of the test; and in contrast to the intense theoretical discussion following its introduction during the 1910's, many psychiatrists were now content to accept the practical consequences of therapy without worrying about the discrepancy of laboratory findings: 'The most important consideration is that the patient must be treated, not the spirochaete or the actual or presumed symptoms of its activity in the central nervous system ...'[46]

The majority of doctors actually viewed the best results of malaria therapy as 'remissions' of GPI; but this description too was open to objection. During early experiments, some doctors claimed that GPI was prone to remissions anyway, and that there was therefore nothing new in witnessing these so-called recoveries. Some extended this argument by suggesting that the reason for a higher frequency of remissions might lie less in the effect of malaria than in a change in the characteristics of GPI. The disease, they pointed out, had become less aggressive and more

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[45] 'General Paralysis: A Discussion ...', 1929, p 295.
[46] Quoting G Holmes, Meeting of the Medical Society for the Study of Venereal Diseases: 'Diagnosis and Treatment of Neurosyphilis', Lancet, Jan 19, 1924, pp 131-2.

amenable to treatment over the past ten to twenty years: perhaps because of the modern arsenical treatment of syphilis which had altered the course of tertiary forms of syphilis; perhaps due to a recent decline in alcohol consumption which had previously masked the symptoms of true GPI. The debate between Brander and the MPA - described in the previous chapter - linked three themes: the identity of GPI, the reliability of laboratory science, and the efficacy of malaria therapy. Brander argued that the perceived change in the characteristics of GPI was not natural, but due to a redefinition imposed by the advent of laboratory science: the Wassermann test was being used to falsely broaden the identity of GPI (to include, for example, other forms of cerebro-spinal syphilis) and so to substantiate claims that higher numbers were amenable to treatment. GPI was, Brander claimed, by definition untreatable; therefore cases in which malaria was supposed to have produced remissions could not in fact be GPI.[47] Although his opinion did not represent the majority, this tortuous debate demonstrated that the new treatment was to some extent stirring up old definitions of GPI; it was, after all, 'the very refractoriness of ordinary anti-specific measures in the pre-malaria treatment days that often confirmed the diagnosis of the paretic...'[48] These concerns led psychiatrists such as Nicol and Hutton to suggest that the diagnosis should be re-established upon sound clinical grounds, in order to judge the true value of the treatment.[49]

There continued, then, to be wide variations in the terms used by British psychiatrists to classify improvement. Although statistical results were often given by

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[47] 'General Paralysis ...', 1929, pp 1 - 10.
[48] W D Nicol and E L Hutton, 'Some Clinical Aspects of General Paralysis', J Ment Sci, 1935, 81, pp 804 - 821; on p 815.

the categories 'discharged', 'improved mentally', 'improved physically', 'no improvement', 'death', the types of patients included under each of these terms differed enormously.[50] Patients discharged or termed 'cured' could range from those still heavily incapacitated and reliant on the care of relatives, to those able to take up their former lives and jobs again. Psychiatrists were often candid about this: 'As to what constituted 'recovery' ... it varied a good deal, depending on the conservatism or the liberalism of the observer's mind ... Some [discharged patients] might keep clear of acute symptoms and appear harmless; some showed a degree of initiative. All, however, showed some feeble-mindedness ...'[51] The Claybury doctor Rudolf agreed: 'An acute observer will notice an improvement where a less acute observer will fail to do so ... A decision that a patient is fit for discharge made by one observer may not be agreed with by a second observer. The social circumstances of a patient may also affect his discharge ...'[52] A stark indicator of these arbitrary definitions was provided by the reported remarks of the Canadian psychiatrist Slight in 1935: '...With malaria therapy [he commented] much could be done in very many cases. It was true that the changes might be mild; the patient might not afterwards be able to initiate new ideas, he might not have the same artistic sense as before, but, as psychiatrists, those present

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[49] Ibid.
[50] This is demonstrated by comparing published results of the Horton with an examination of clinical records. My own interpretation of different categories - from descriptions of clinical outcomes - differed considerably from those of the published categories.
[51] W D Nicol, 'The Treatment of General Paralysis by Malaria', Brit J Ven Dis, 1929, 5, 2, pp 85 - 109; on p 109.
[52] 'General Paralysis ...', 1929, p 20.

were not interested in these fine differences of character. From the clinical standpoint one could get 100% recoveries.'[53]

Variations in the standard of improvement between observers were not the only obstacles to assessing results. Rudolf also mentioned differential selection of cases for treatment (healthier patients were more often selected), variations in the size of series, differences in treatment protocol, and 'other factors, largely unknown, collectively termed "chance"'.[54] All of these factors were continually acknowledged, but there was no serious attempt to introduce standardised criteria. Often their importance was minimised, on the basis that pragmatism was of more interest than experimental rigour. Rear-Admiral Meagher, for example, whose report I shall return to shortly, acknowledged that selection of cases for treatment did occur, but pointed out that the profession and the public were only interested in the number of discharges achievable, not the initial condition of these discharges. It was such social pragmatism - which pervaded published studies and clinical case-notes - that finally offered the strongest perception of the benefits of malaria therapy. A blunt definition by the Medical Officer of Cheddleton Asylum demonstrated this: 'The standard set for cure or great improvement is that the patient should either return to his proper work outside or carry out intelligent and valuable work in hospital. "Improved" implies manual labour in hospital - the condition of a good working dement. "Not improved" includes non-workers and the dead.'[55] Follow-up studies of discharged patients often concentrated upon renewed wage-earning capacity as a standard of

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[53] Nicol and Hutton, 1935, p 819.
[54] 'General Paralysis ...', 1929, p 19.
[55] Quoting F H Stewart, Ibid, p 18.

recovery or - in the case of women - ability to care for their family again. The superintendent of Parkside Asylum in Cheshire, for example, sent a simple (and self-confessedly crude) questionnaire to nineteen patients discharged over six years (representing 35% of the treated sample), asking: 1. Are you keeping in good health? 2. Are you in employment? 3. If not in employment, is it due to your health, or to inability to obtain work? Fourteen, he reported, were in good health, and nine in employment.[56]

If not suitable for discharge, a treated patient might demonstrate improvement by useful work within the asylum. Clinical records were filled with such descriptions of treated patients: '[Successfully treated] patients are marked by their willingness to assist in ward work and later out-of-doors ... they become hard-working ...'; '[She is now] childish and garrulous, but a good kitchen worker'; 'Better behaved socially; she employs herself usefully'; 'As a result of her malarial treatment, the progress of her GPI has been completely arrested. Usefully employed, quiet, cheerful, happy ...'[57] Short of rendering them useful, malaria therapy could make patients more manageable by improving their behaviour and their control over bodily functions. The 'filthy habits', or 'wet and dirty habits' - predominantly soiling - of the general paralytic were usually spoken of in admonitory tones by doctors; and in this sense malaria

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[56] H D Cormac, 'The After-History of Treated General Paralytics Discharged from Parkside', published in Eighteenth Annual Report of the Board of Control for the Year 1931 (HMSO: London, 1932), p 128.

[57] Clinical case-books of Horton Hospital for 1929. There are numerous similar examples; see for example G E Shand, 'Old and New Methods of Treatment in General Paralysis: A Comparison of Results', J Ment Sci, 1929, 75, pp 250 - 256; Yorke, 1926, p 428: 'In a disease like general paralysis where the mental changes are pronounced, the only result which can be regarded as really satisfactory is that in which the remission is so complete that the patient regains his mental and physical powers and is enabled to return to his occupation and once more become a reasonable and useful member of society ...'

therapy sometimes took on an uneasy identity as moral corrective as well as physical treatment: '... [She] gives us no active trouble, is quieter and better behaved socially ... cleaner in habits ...' [58] I would question whether this was peculiarly associated with the issue of syphilis - as Braslow suggests.[59] Such statements could equally apply to any patient in British asylums: malaria was simply a seemingly powerful tool, which could improve the behaviour of the unmanageable insane. Many doctors saw this as complete justification for using the therapy - whatever the discharge rates might be.

The Dangers of Malaria

It was well recognised that malaria therapy could cause potentially dangerous side-effects ranging from jaundice, muscle pains, weight loss, herpes, gastro-enteritis, exhaustion and anaemia to delirium, convulsions and death. Less serious, but as worrying for patients, were malaria relapses which commonly occurred about six months after treatment in those infected with mosquitoes. Acute side-effects seem to have occurred in most patients: one or more, for example, was documented in practically every patient entered in the Horton clinical records from 1925 to 1927. These curt clinical descriptions gave some sense of how harrowing the procedure was; and they also stressed the importance of the doctor's observation and judgement in attempting to balance the well-being of the patient with the efficacy of his fever. As Yorke pointed out: 'In the hands of the inexperienced the malaria treatment is undoubtedly a two-edged sword ... The decision as to when to end the febrile attacks

[58] Clinical case-books of Horton Hospital for 1929; see also, for example, N B Graham, 'The Malarial Treatment of General Paralysis', J Ment Sci, 1925, 71, pp 424 - 431; on p 426: 'The patient's filthy habits are controlled'.

by quinine treatment is the chief cause of anxiety to the practitioner; if the febrile attacks be stopped prematurely no good may result, and if they be allowed to continue too long the patient's life may be sacrificed.'[60]

Most published reports included in their statistics the number of such sacrifices occurring in the treatment group - although with variable attempts to differentiate between those directly attributable to malaria, and those attributable to intercurrent illness or to GPI itself. In nine of the first asylums publishing results, deaths were reported in 14 to 32% of patients: these usually included deaths occurring up to one year after the treatment; and proportions ranging from 5 to 15% were considered probably due to the treatment itself. Whether these figures were faithfully reported we can only speculate: a report from America in 1925 suggested that the number of deaths attributable to malaria were not being acknowledged: '... Malaria probably hastened the end in other studies where it was claimed that intercurrent disease killed the patient ...'[61]

The attitudes of doctors towards these considerable risks were by no means straightforward. Those who experimented early with the treatment betrayed a rather cavalier attitude - which resulted in noticeably higher numbers of deaths than were reported on the Continent. During 1923 the German Professor Muhlens raised concerns about British practice in a letter to the Journal of Mental Science, drawing attention to an unacceptable number of 'unfortunate accidents' and deaths: '... We have frequently pointed out the dangers of this treatment when improperly applied ...

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[59] Braslow, 1996.
[60] Yorke 1926, p 429 and p 425.

The treatment ... is by no means to be entrusted to all neurological physicians as a general therapeutic measure'. He offered guidelines which, he hoped, would '... avoid bringing into disrepute a therapeutic measure which I consider the best method of treatment for general paralysis'.[62]

The response of the Board of Control and Ministry to these concerns set a pattern for the following twenty years. Colonel James, as I have described, played a large part in alerting the Board to the potential dangers of treatment. His first report from the Continent stimulated the Board to enquire about current practices in asylums during 1923; and his concern that early strains of malaria had a high tendency to cause complications and death - highlighted by Mühlens' letter - led to the 1924 circular which advised psychiatrists upon precautions to minimise risks. The Board was keen to advertise the fact that it, and the Ministry of Health, was taking an active and responsible role in monitoring and controlling the dangers arising from the new treatment. Copies of the circular were published in the major medical journals, in order to show 'that both departments have been wide awake over the business and are taking good common sense lines to secure precautions as well as progress.'[63] There were repeated discussions in the literature over the following years about the need to manage patients with care, observe precautions, and select only those who were fit enough to withstand the fever. When, for example, the 1925 annual statistics showed that, of 190 patients who had died of GPI during the year, no less than 91 related to

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[61] G W T H Fleming, 'Lewis, Nolan, D C: The Present Status of the Malarial Innoculation Treatment for General Paresis', J Ment Sci, 1925, pp 605-8.
[62] See P Mühlens, 'The Dangers of the Fever Treatment of Paralysis', 1923, transl in J Ment Sci, 1924, 70, pp 340-1.

malaria treatment, the Board stressed that cases should be grouped according to the physical stage of illness to minimise risk.

But there were clearly limits to the Board's involvement with day-to-day asylum practice, illustrating a somewhat 'hands-off' approach which Fennell has described as characteristic of the period.[64] It is perhaps surprising that after circulars in 1924 and 1926, no further advice was circulated to asylum superintendents; indeed many remained unaware of the facilities that were available to them for the institution of treatment.[65] Concern to provide purer and safer strains of malaria was stressed as the major stimulus to developing the Horton treatment centre and laboratory in 1925; but the Horton strain was simply on offer - there was no obligation to use this material for the inoculation of patients. Although Commissioners clearly had the responsibility of checking the application of their guidelines in individual asylums, there is no evidence as to how far they were enforced. The issue of consent illustrated this lack of involvement clearly. During the earliest experiments, doctors made individual decisions as to whether consent was necessary, and practice varied according to their perception as to why it might be indicated. In answer to an early enquiry, for example, a psychiatrist at the City of London Asylum replied: 'During my absence from home the cases were inoculated without communicating with the friends but I have given directions that in all cases in future, the consent of the friends must be

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[63] Letter from Buchanan (Board of Control) to the Ministry of Health, 3 March, 1924. PRO, Kew: MH 51/698.
[64] Fennell, 1996, pp 129 - 150.
[65] The Board of Control regularly received enquiries from asylum superintendents, as late as the 1940's, asking how to obtain supplies and institute treatment.

obtained first ...'[66] In contrast the superintendent of Whittingham Asylum admitted: 'To begin with consent was always asked of relatives and almost always readily given - latterly I have not considered this necessary as malaria treatment at present may be said to be the usual and recognised treatment for GPI on the Continent at any rate'.[67] Although the circulars carried no guidelines concerning consent, the Board occasionally made its opinion known in letters to individual asylums: '... It is probably early days to assume consent of the relatives and ... if it is not actually asked, it would be a desirable precaution to acquaint them of what is proposed, giving time for them to send in an objection. Indeed, should good results become much more certain it may even then be desirable because of the fact that the patients are under detention and one is actually inoculating them with a disease which is occasionally swiftly fatal - in order to protect yourself and your Committee from the unpleasantness of an action at law ...'[68] Subsequent practice is difficult to assess, since there was rarely any indication in clinical notes as to whether consent had been obtained; however it appears to have become more standard practice over time. In a 1937 report the Board stated more dogmatically that they would not interfere with the use of experimental treatment by medical superintendents '... so long as the written consent of the patient's nearest relative has been obtained'.[69] It is equally difficult to gauge the responses of patients' relatives to this issue. One instance of a man objecting to the treatment of his wife by malaria was reported and discussed by the

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[66] Letter from Keen to Board of Control, 11 June 1923. PRO, Kew: MH 51/697.
[67] Letter from Clarke to Board of Control, 13 June, 1923. PRO, Kew: MH 51/697.
[68] Letter from Bond to Clarke, 25 June, 1923. PRO, Kew: MH 51/697.
[69] D McRae, 'Presidential Address at the 96th Annual Meeting of the Royal Medico-Psychological Association, 1937: Some Observations on the Care of the Insane', J Ment Sci, 1937, 83, pp 489 - 504;

Sub-Committee of the LCC in 1929, suggesting that this was probably an unusual occurrence.[70]

Fennell claims that consent was a non-issue for psychiatrists during the period (apart from areas such as surgery and sterilisation); and that the right to treat detained patients at medical discretion was the paramount consideration.[71] The response to malaria therapy does not completely bear this out, since both asylum doctors and the Board were clear that consent was desirable for several reasons: because of the possibility of legal redress; because the treatment was still arguably in the experimental stages; and because of the risks involved. Nevertheless there was little enthusiastic enforcement; and it is impossible to tell from available records how far consent was practised; or how fully the treatment was explained to patients or relatives.

After the early situation in which the Board of Control had found itself catching up with experimentation which had already started in asylums, the dynamics between Board and Ministry, and working psychiatrists changed to a certain extent. Certainly many ordinary doctors expressed a pragmatic acceptance of the risks of malaria therapy, and deaths continued to be reported as a matter of course, with little comment. GPI after all was a hitherto hopeless disease with a death rate of 90%, and risks seemed an acceptable price to pay for the evident benefits: '... With a deadly disorder of this kind, it can be said to have achieved results sufficient to justify its use

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p 499.

[70] Report of the LCC, 2 Aug, 1929. GLRO: LCC/MIN 1149.

[71] Fennell, 1996.

until some other less drastic method is found ...'[72] But increasingly the Board perceived strains of resistance to implementing the treatment. On the one hand James continued to act as its 'conscience', repeatedly drawing attention to the evident dangers. In a 1927 Ministry of Health report he pointed out that of 543 patients treated during the previous year, 117 (21%) had since died - half within six weeks of the fever inoculation.[73] These statistics, he warned, threatened to bring discredit upon malaria therapy. He asked that cautionary procedures should be re-circulated to all of the asylum Medical Officers - a request which caused a small flurry of correspondence between the Ministry and the Board. The report was disquieting, the Ministry noted, and suggested that closer supervision was needed; however it would not be expedient to include the figures in the Board report, since such a step might cause panic amongst psychiatrists and jeopardise the continuation of trials. Malaria therapy, it reaffirmed, was the best chance for GPI patients, and must be fully assessed. The Board agreed; and the figures were duly omitted from reports.[74]

The Ministry and Board felt that they had good reason to be economical with the truth. Uptake of malaria therapy in asylums was respectable but undramatic, and the authorities detected a certain reluctance which they attributed partly to concern about the treatment's dangers. In 1923 16 out of the 98 public asylums were using the treatment, as well as the registered hospitals of Bethlem and Camberwell. These figures rose to 24 public and 8 registered hospitals in 1924, and 32 and 11 respectively in 1925 - by which time it was reported that 896 patients in total had

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[72] G Riddoch, 'Neurosyphilis', Brit J Ven Dis, Jan 1928, 4, 1, pp 1- 13; on p 5.
[73] Report by James, May 1927. PRO, Kew: MH 51/698.
[74] See memos by Carnwath and Willis attached to James' report, ibid.

been treated.[75] To many this indicated a disappointing lethargy on the part of British psychiatrists. This was compounded by disappointment that discharge figures were not as spectacular as claims from the Continent. Despite the improvement of British results, so that the average discharge rate settled at 30%, the perception lingered that the Europeans did better. This discrepancy could be explained away by pointing to continental clinics which had larger pools of patients, so making experimentation easier; to earlier treatment owing to wider facilities for voluntary care; and to exaggeration of reports - a symptom of European enthusiasm!

The British, however, clearly had to hold their own; and the response of the Board and the Lancet was to vigorously endorse a full trial of the therapy: 'Until recently general paralysis has been regarded as an incurable disease ... and a feeling of helplessness was engendered ... Any therapeutic measure therefore that carries with it a glimmer of hope must be accepted at its face value, and tested to the utmost limit of possibility, in order that a correct estimate of its utility may be formed.'[76] They constantly stressed that dangers of treatment were justified by the severity and hopelessness of GPI. The reality of risk, indeed, was often integral to heroic descriptions of the therapy: 'Malaria therapy gives remarkable results ... The mortality of induced malaria is still fairly high ... On the other hand, the disease to be treated is one which makes big risks justifiable.'[77] In 1925 a Lancet leader complained that more hospitals had not tried the treatment, and suggested possible reasons for this apparent lack of enthusiasm. The popularly recognised British

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[75] Report by Board of Control, July 1927. PRO, Kew: MH 51/538.
[76] Eleventh Annual report of the Board of Control for 1924 (HMSO: London, 1925), p 103.
[77] Leader: 'Pyrexia in the Treatment of GPI', Lancet, Aug 20, 1932, pp 406-7.

caution towards scientific innovation bore the brunt of the blame; psychiatrists were evidently uneasy about the dangers and dubious benefits of a treatment that had not been proven. Nonetheless, the article urged that doctors should throw caution to the wind, and make a proper trial of the therapy: 'British psychiatrists are still rather suspicious of a method so drastic and empirical ... [But] ... Desperate ills demand desperate remedies; there is very little danger in the treatment to patients of ordinary stamina, and so far no other means has been offered of countering one of the most distressing and hopeless illnesses of our age.'[78] During the 1920's the Board commissioners continued to make exhortations in their rhetoric, and to politely admonish backward asylums during their visits of inspection. Board reports were full of such comments: 'We hope serious consideration will be given to instituting malaria therapy ...'; 'It is such a pity that patients are missing their chance of this treatment'; 'Malaria treatment is desirable for such a dire disease ... other asylums are taking it up ...'[79]

Confirmation of individual psychiatrists' misgivings is hard to come by. Replies to a survey contained in the Board's 1924 circular indicated that several superintendents thought the treatment was too dangerous, and should be instituted only when a proper trial had been carried out; but how many doctors hesitated specifically because of this is unclear. Sometimes individual doctors expressed concern about the treatment, such as Dattner, of the National Hospital Queen Square, who was quoted as '... having had very unfortunate experiences with malaria ... I was

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[78] Leader: 'The Malarial Treatment of GPI', Lancet, Aug 22, 1925, pp 389-90.
[79] Extracts from individual asylum inspection reports, Fifteenth Annual Report of the Board of Control for 1928 (HMSO: London, 1929).

very glad to avoid using it ...'[80] The psychiatrist Shaw expressed humorous unease during an MPA discussion in 1929. He imagined a future historian commenting upon this episode in medical history and concluding that - as contemporary doctors must have realised that there was no biological antagonism between syphilis and malaria - the explanation must lie in 'an organised attempt to deplete their insane population by infecting them with malaria ... [It] would undoubtedly result in increased mortality amongst the debilitated insane'[81] The psychiatrist Menzie concluded the discussion with an image of malaria therapy as a sword of Damocles hanging over the profession and their patients: '... With regard to what [the President] had said about malarial therapy, the old Biblical phrase occurred to him, 'Had Zimri peace, who slew his master?'[82] Such comments were rare; but it is quite possible, of course, that those who objected to the therapy tended not to have their views published in the journals - which therefore give a false impression of approval.

There were, however, other possible reasons for the sluggish uptake of malaria therapy. The Board's 1924 survey revealed that many doctors were hindered for practical reasons: no suitable cases had arrived; or the hospital had inadequate laboratory facilities. 'Unsuitable' cases were generally patients admitted to public asylums who were considered too advanced in their disease for treatment; this problem led to repeated calls for the earlier detection of GPI, so that malaria therapy could be instituted earlier when it seemed more effective. Lack of laboratory facilities should have been a less intractable difficulty. Since the early twentieth century,

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[80] Quoted in Purdon Martin, 1972, p 160.
[81] 'General Paralysis: A Discussion ...', 1929, p 16.
[82] Ibid, p 297.

commissioners had anxiously watched the development of the scientific approach in asylums, and had tried to monitor the extent of laboratory facilities for routine and research work. The 1922 annual Board report noted that clinical laboratories now existed in 'a considerable number of mental hospitals, but by no means all of them', repeating - as many times before - that no mental hospital should be without such facilities.[83] As part of their drive to foster scientific work in asylums, they began to publish in detail research abstracts and reports of routine laboratory work from any asylum who cared to submit them, necessitating a large second section to the annual reports. During the 1920's there was steady progress in the accumulation of laboratories until, by the 1930's, about 80% of asylums had such facilities, and a similar percentage were regularly sending the Board reports of their scientific work. As was the case with the Wassermann test itself, the availability of laboratories doubtless did correlate to some extent with the institution of malaria therapy. During a discussion of the MPA in 1929, for example, the superintendent of Norwich asylum noted that he had no laboratory at his disposal, and worked with an insufficient staff to experiment; he suggested that the new tryparsamide treatment might be more suitable for those in a similar situation to himself who nevertheless wished to make a scientific contribution to treatment.[84] Reflecting this link, the Commissioners' chidings about malaria therapy were usually made in the same breath as those about laboratory facilities and research; despite its lack of theoretical clarity, the therapy was regarded as very much part of the scientific spirit that asylums should be

[83] Ninth Annual Report of the Board of Control for 1922 (HMSO:London, 1923), p 56.
[84] 'General Paralysis ...', 1929, p 283.

fostering. As the Horton Centre and improved methods of transport became established, the excuse of poor laboratory facilities appeared less convincing - as Rudolf's retort to the Norwich doctor quoted above demonstrated: 'All that one needed were a microscope, slides, and Leishman's stain, now that blood or mosquitoes could be sent anywhere in the British Isles ...'[85] A further speaker confirmed: 'Few places were more remote from civilisation than Western Argyll, yet malarial blood had been conveyed successfully down there, a thermos flask being used ...'[86] Nevertheless, it remained clear that far fewer asylums were using the treatment than had access to adequate laboratory facilities.

Surgeon Rear-Admiral Meagher's report

By 1927 nearly 1600 general paralytics had been treated with malaria, and the Board of Control commissioned a large-scale analysis of results intended to judge the benefits of malaria therapy, 'for the satisfaction of both professionals and the general public'.[87] The task of analysing the data fell to the senior medical commissioner Surgeon Rear-Admiral Meagher. His study, published in 1928, was the first large-scale attempt to follow up patients several years after their discharge, and compared outcomes in all those treated between 1922 and 1927 with all those untreated.[88] Information was gathered through forms sent to asylums, and through home visits and assessments. Meagher was at pains to demonstrate that he was not painting a falsely

[85] 'General Paralysis ...', 1929, p 285.
[86] Ibid, p 288.
[87] Thirteenth Annual Report of the Board of Control for 1926 (HMSO: London, 1927), p 84.
[88] This form of comparative enquiry was made on the recommendation of Major Greenwood, the Medical Statistical Officer: 1597 treated patients were compared with 1173 untreated patients.

glowing picture of the therapy. His descriptions of treated patients appeared to be rigorously honest, and his conclusions were full of cautions and provisos. His analyses were simple, and he stressed throughout his report that confounding factors could not be entirely eliminated, and that a rigorous statistician might well have objections to his methods.

Despite all this, he succeeded in producing a report which epitomised the philosophy of 'benefits outweighing risks' which the Board was so keen to foster. His oft-quoted conclusion maintained that malaria therapy had proved its worth, and could be claimed as the 'treatment par excellence' for GPI: 'It appears to have been definitely established that malarial treatment increases the length of life, renders existence more natural, and produces improvement in the physical condition and mental state. In several instances complete recovery seems to have been achieved, and the treatment offers more promise of success than any other form that has been given extended trial.'[89] 26% of treated patients, he reported, were now surviving outside asylums, compared with 2% untreated. Of these, roughly one third were apparently 'fully re-established'; one third had arrested disease which left 'minor traces'; and one third still had significant disease - although they had benefited from the treatment. The majority were following their usual occupation. 32% of treated patients were alive in hospital, compared with 8% untreated; of these roughly 40% showed improvement and a further 25% showed arrest of their disease. '[They] lead a more desirable existence ... are more amenable, require less supervision, and retain

[89] E T Meagher, 'General Paralysis and its Treatment by Induced Malaria', J Ment Sci, 1929, 75, pp 714-7; on p 717.

more physical strength than other general paralytics. They are cleaner in habits and less degraded ... Nurses who have spent many years in hospital service ... expressed themselves enthusiastically concerning the improved general existence of the treated cases ...'[90] Finally 42% were now dead, comparing favourably with 90% of untreated patients.

Meagher was evidently convinced that malaria treatment had a beneficial influence; but his full report in fact gave a far more mixed picture than the optimistically worded conclusion implied. He noted that, whilst return to employment was usually taken as the criterion of 'cure', such patients usually had erratic behaviours suggesting residual or latent mental disease. He compared such patients to the alcoholic who comes to his senses at dawn: '... He now appreciates the foolishness of his former optimisms ... judgement and insight have been restored ...' Nevertheless, 'although a good number are found with little or no mental disorder remaining ... nervous signs may persist and the facial expression may denote anxiety ... there may be twitching of the facial muscles, he is readily startled or confused, gets tired quicker than he did, reads little, and is apt to shun society and to remain more at home than was his wont ... Frequent mention was made of the irritability which the recovered case displays, particularly of his intolerance of the noise of his children ... It was found better, too, not to enter into argument with him. He was too readily huffed ...'[91] A smaller group of dischargees were fit for little or no work: '...Their judgement is not too reliable, their horizon is limited, they are, perhaps, a little too

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[90] E T Meagher, General Paralysis and its Treatment by Induced Malaria (London: HMSO, 1929), p 48. PRO MH 51/537.
[91] Ibid, p 52.

satisfied, and there is a slight ... dullness of mentality ...' Finally, some of the dischargees '... might have been better left certified ...' Meagher noted the struggle for survival in many families of 'recovered' general paralytics, and the frequent marital discord. The remark, he documented, had been made on more than one occasion: 'Why not let them die?'. He stressed that these observations had to be included in the interests of honesty: 'In rendering an account of these discharged cases, the object has been to portray them as they appealed to the observer and to avoid anything which might in the least show them as being better than they really were ...'[92] Indeed, he conceded that his own results often compared unfavourably with the claims of the asylums themselves.

The Board, despite earlier qualms, made no attempt to suppress Meagher's observation of an 'exceptionally high death rate in the first and second month after malaria therapy ...'[93] Whilst acknowledging that malaria was a risky treatment and that 'its administration should be attended with unremitting care ...', Meagher definitively justified the risks. Many of the deaths, he pointed out, were probably due to poor selection of cases; Austrian clinics, in contrast, claimed extremely low death rates because of rigorous attention to the state of the patient before treatment. He concluded: '... Sympathy may be extended to those who feel over-caution to be a mistake. Desperate evils and desperate remedies are proverbially associated; and if, after adequate explanation, consent is given, need there be greater hesitation in applying this remedy than in attempting a surgical operation as the one and only

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[92] Ibid.
[93] Meagher estimated the figure at a fifth of treated cases; Ibid, p 68.

chance of saving life when the chances are equal that the patient may die on the table?'[94]

Despite the unfavourable parts of Meagher's report, his positive conclusion was seized upon by the Board as confirming the optimism of the past seven years. The report was prefaced by the commissioner Sir Hubert Bond, who wrote a detailed and triumphal history of GPI and the advances that had been made since the turn of the century: malaria therapy - now vindicated by a proper trial - was portrayed as the culmination of these advances. There was a certain excitement over the report in Board and Ministry circles. The commissioner Brock wrote: 'I do want to make it clear that this is really a big thing - perhaps the biggest thing in the way of active treatment of any form of insanity which has been discovered yet'.[95] The study was regarded as a turning-point: malaria therapy had successfully passed its probationary period, and it was confidently predicted that there would now be a sharp increase in the hitherto disappointing uptake of treatment. Malaria therapy could now be portrayed as the only bright spot amidst the general therapeutic gloom: the Board's annual report for 1929 lamented the fact that there had been little change in the recovery rate for the majority of mental diseases over the years - but noted that there was now 'one outstanding exception - malaria therapy'. This was, it declared, 'a matter of great moment ...'[96]

The report had implications, too, for the Horton hospital. It was now felt to be crucial that the centre should be expanded to fulfil the roles of research, mosquito

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[94] Ibid, p 27.
[95] Letter from Brock to Leggett (Ministry of Health), April 1928. PRO, Kew: MH 51/698.
[96] Fifteenth Annual Report of the Board of Control for 1928 (HMSO:London, 1929), p 2.

supply and training for the country: the fact that the treatment was both a major advance and potentially dangerous necessitated investment in this centre of excellence. By 1928, the Ministry and the Board were discussing the addition of a male ward and laboratory expansion, as the preliminary to an official scheme of malaria treatment for the whole of Great Britain. A dispute, however, arose between the LCC and the Ministry of Health as to who should continue to fund the Horton - and in particular the proposed expansion. Meagher's report was pivotal to this dispute, since if the treatment was no longer experimental but established, the Ministry argued, it was no longer their province to fund it, but that of the local authority and the Board of Control. The latter, reluctant to provide resources, continued to argue that Horton could still be regarded as an experimental centre, stressing its active role in supplying and educating other hospitals, and avoiding the dangers implicit in malaria treatment. This deadlock over funding continued for several years until, in 1931, the Ministry agreed to continue to provide funds for the Horton facilities until 1935. A year later, male patients were admitted for the first time.[97]

Into the 1930s: Psychiatry and Malariology

By the 1930's, accounts of malaria therapy no longer possessed their former dramatic interest. Meagher's study had appeared to mark the treatment's transition from experimental to established procedure; nevertheless, the optimistic interpretation of

[97] See report of meeting between Board of Control and LCC, May 1928; correspondence between Leggett and Brock, April/ May 1928; report by Bond, 9 Dec 1930; report of meeting between Board of Control and Ministry of Health, 13 Feb 1931. PRO, Kew: MH 51/698.

his work did not produce the surge in uptake amongst asylums which the Board had predicted. By 1933 still only about 60% of public asylums were using the treatment; although the record for private hospitals now appeared to be far better - 54 out of 56 reporting that they were using the therapy (although presumably on a far more limited scale than the public asylums). The disappointing uptake was still blamed largely upon doctors' suspicions of the dangers of the treatment. As the commissioner Brock reported in 1933: '... Anything which will improve technique and reduce the mortality rate will help to encourage its adoption in the hospitals which have so far hung back ...'[98] As late as 1944, there was a perception that psychiatrists were still reluctant: one report noted that mortality rate was between 2 and 10%, 'and this knowledge may be responsible for the lack of enthusiasm shown for the treatment.'[99] Again, though, there is little firm evidence of individual misgivings. William Sargant mentioned in 1946 that one 'conservative medical superintendent' had provided the control case of non-treatment by malaria for many years; a comment which suggests that antagonism - certainly after the early period - was rare enough to be noted.[100] Unfortunately there are no figures available indicating the number of asylums using the therapy after 1933 - so that these rather conflicting impressions are not easily reconciled.

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[98] Report by Brock, 3 March 1933. PRO, Kew: MH 51/698.
[99] F G Lescher and H R M Richards, 'Malaria Treatment in the Earlier Stages of Neurosyphilis', Brit J Ven Dis, 1944, 20, 1, pp 37 - 41; on p 37.
[100] W Sargant and E Slater, An Introduction to Physical Methods of Treatment in Psychiatry (Edinburgh: E&S Livingstone Ltd, 1946), p 149.

The stimulus of the Horton centre seemed as important as ever in encouraging malaria therapy amongst other asylums.[101] Increasingly, however, its general research role was overtaking its treatment role in importance. In 1926, the parasitologist Yorke had waxed lyrical upon the fruitful interaction of psychiatry and tropical medicine which malaria therapy had allowed: 'This interesting reaction of one disease upon another has ... a two-fold effect in that it blesseth him that gives and him that takes ... A not inconsiderable proportion of the paretics are restored to health, and those who suffer from malaria will doubtless in due course benefit from the advance of knowledge that the remarkable influence of this infection on the nervous disease alone has made possible ...'[102] Since the establishment of malaria therapy in Britain, psychiatrists had been dependent upon the expertise of specialists in tropical medicine for infected blood and for advice on controlling the induced disease. Individual doctors such as Yorke had quickly grasped this opportunity for research into malaria itself, a fact acknowledged by the Board: '...We note with much satisfaction [that] this opportunity - in some respects unique - is not being neglected by those skilled in parasitology, to enlarge our knowledge of malaria itself; ... facts so ascertained may easily prove to be of widespread advantage.'[103]

Such collaboration was institutionalised at the Horton Treatment Centre, where malaria research, under the leadership of James, went hand-in hand with treatment. James published the first fruits of his research in a League of Nations report in 1926: work which included observations into the natural history of

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[101] See letter from Barter (Board of Control) to Bailey (Ministry of Health): 6 March 1933. PRO, Kew: MH 51/698.
[102] Yorke, 'Malarial Treatment ...', 1926, p 431.

mosquitoes and sporozoites, the transmission of malaria, immunity, and the induced disease in patients. James stressed that this research had arisen simply as a happy by-product of psychiatric treatment: '... The purpose of the laboratory work ... is to fulfil official requirements relating to the treatment of mental diseases ... During the course of the work we have made some observations ... which are important both because they facilitate the work itself and because they can be applied to assist in solving problems of endemic and epidemic malaria in nature ...'[104] His obituarist again stressed this opportunism: '... By a happy inspiration of its originator, the Centre has been made a means of carrying out scientific investigation upon malaria ... which has greatly extended our knowledge of the disease.'[105]

Yet it soon seemed that malaria research - with its colonial interest - was overshadowing psychiatry at the Horton. Meagher's report - describing the successes and dangers of malarial therapy itself - had been the main bargaining point in negotiations concerning funding of the centre during the early 1930's. By 1935 when the question arose again, however, there was far more emphasis upon the wider benefits of the malaria work. The LCC, urging the Ministry of Health to renew their support, wrote: '... The work in connection with this experiment has enabled most valuable research into the nature and treatment of malaria to be undertaken which has yielded results of great imperial and international importance as well as being of considerable assistance to the Ministry in its own sphere of preventive medicine. The research work is .... such as to affect not only the work of this Ministry and the Board

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[103] Twelfth Annual Report of the Board of Control for 1925 (HMSO:London, 1926), p 98.
[104] James, Report on the first results of laboratory work ... (1926), pp 4 and 27.
[105] Christophers, 1947, p 515.

of Control but also has considerable interest and value to the Colonial Office and the MRC.'[106]

Over the following years, the Horton was the site of much influential malaria research, including the identification of the pre-erythrocytic parasite in man and the testing of synthetic anti-malarial drugs; and work such as this became increasingly important with the advent of the Second World War.[107] The Centre gained a strong reputation throughout Europe: staff were able to boast that Wagner-Jauregg himself had adopted the Horton technique for examining blood films; and that the Horton insectarium plans had been copied throughout the Continent. It was headed, too, by a succession of important figures in malariology: James himself (who retired in 1934 but remained involved until his death); John Alexander Sinton; Gordon Covell; PCC Garnham; and Shute, who was associated with the Laboratory for most of its fifty-year history.[108] The benefits of the collaboration were not limited to the Horton: James, discussing recent advances in his field in 1937, spoke with satisfaction of 'the establishment in mental hospitals where malariotherapy was practised of research labs charged with the duty of cultivating malaria parasites in mosquitoes ... This has opened up an entirely new field of research...'[109]

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[106] Letter from Leggett to the Treasury, 24 June 1935. PRO, Kew: MH 51/698.

[107] See Rollin, 1994, pp 94-7. The Centre, for example, fostered close co-operation with the Bayer chemical factories at Leverkusen and research laboratories at Eberfeld, through its research into anti-malarial drugs.

[108] J R Glynn's thesis on the relationship between dose and disease virulence includes a study of the records of malaria therapy at Horton; it thus gives some insight into the kind of malarial research which took place during the 1930's: J R Glynn, 'Studies on the Influence of Infecting Dose on the Severity of Disease', (Unpub doctoral thesis: London School of Hygiene and Tropical Medicine, 1993). Extensive records of P G Shute's work and correspondence are held at the Wellcome Institute for the History of Medicine.

[109] S P James, 'Advances in the Knowledge of Malaria: Presidential Address at the Royal Society of Tropical Medicine and Hygiene', BMJ, Nov 13, 1937, p 989.

But whilst this high-profile research attracted international interest and resources, research into GPI itself was regarded as less impressive; many doctors, indeed, appeared to remain unaware of the treatment facilities available. A report in the Lancet of 1937 drew attention to this, noting the achievements of the centre in terms of parasitology: '... A matter of great importance in view of the close and constant relations between this country and other parts of the empire where malaria is endemic'.[110] Nonetheless, it continued: 'Those responsible for the mental hospitals of the London County Council are anxious that the opportunities afforded at the Horton for the treatment of neurosyphilis and general paralysis should be better known ... A large unexplored field of research relating to the problem of neurosyphilis is open to workers, who should be attracted to the centre at Horton, just as during the past 10 years malariologists have been attracted.'[111] This discrepancy put into perspective any triumphalism that psychiatrists might have felt about malaria therapy: tropical medicine simply over-shadowed psychiatry in terms of excitement and therapeutic optimism. The more conspiratorial view, that psychiatric patients were being used as objects of research, is hinted at by Fennell: 'Whatever its efficacy, the use of [malaria therapy] also provided a ready pool of human subjects for research purposes, giving the opportunity to gather information on the disease process of malaria in controlled circumstances.'[112] Such a view can only be a matter of interpretation - although it is worth pointing out that the bulk of malaria research was carried out upon mosquitoes in the laboratory. Beyond this, whilst it seems that malaria research did arise simply

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[110] 'A Malarial Treatment Centre: The Horton Unit', Lancet, May 22, 1937, pp 1081-2.
[111] Ibid, p 1082.
[112] Fennell 1996, p 126.

as a by-product of psychiatric treatment, it is also probable that thereafter it provided an intellectual and practical stimulus for continued interest in GPI.

Before the Asylum: Early Treatment of Neurosyphilis

During the inter-war years psychiatrists increasingly turned their attention to illness beyond the limits of the asylum. Long-voiced pleas for preventive psychiatry and adequate after-care were translated into practice with varying degrees of enthusiasm and success through the establishment of out-patient clinics and through the development of the social work profession; and the 1930 Mental Treatment Act provided further impetus to this attempted move into the community.[113] The Wassermann test had already offered doctors the opportunity to take advantage of these trends by broadening the definition of neurosyphilis and undertaking surveillance of those with very early neurosyphilis. I have suggested, however, that there was probably a considerable gap between hopes expressed in this area and achievements in practice. Malaria therapy offered a further opportunity for psychiatrists attempting to broaden their role: not only did it appear applicable to the early stages of GPI; it also resulted in a sizeable population of treated ex-asylum patients who were potential targets of follow-up and support.

The putative starting-point of neurosyphilis had been pushed to ever earlier stages during the later nineteenth and early twentieth centuries. As the tools of laboratory medicine - both the Wassermann test and a variety of CSF analyses - were applied to the disease, doctors claimed that its seeds were sown even during the first

few weeks of a primary syphilis infection. Initially, the most important implication of this had been in the surveillance of patients; early treatment of neurosyphilis was less attainable, since the only therapeutic option was an intensification of treatment for syphilis itself. The arrival of malaria therapy, however, made early neurosyphilis a potentially more practical problem, since it was accepted that the earlier a case was treated, the more effective was the therapy.[114]

During the 1920's, certification procedures placed a firm constraint upon how early a case of GPI could be treated. Since any asylum patient had to be certifiable - and therefore in a relatively advanced stage - the only possible sites for early treatment were general hospitals, private hospitals, or patients' homes. It is difficult to tell how widely these options were taken up before and after the 1930's. Home malaria treatment was rarely reported, and one can assume that it was hardly ever performed due to the accompanying difficulties and dangers; nonetheless the Board of Control did not appear to have any strong objection to the practice.[115] Licensed and registered hospitals used malaria therapy in increasing numbers from the early 1920's and might have been expected to treat uncertified patients; however, relatively few GPI patients were accepted by them in the first place, and, besides, results were not recorded. General and specialist hospitals, too, apparently treated increasing numbers

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[113] Jones, 1993, pp 126 - 140.

[114] See, for example, W M McAlister, 'The Role of Infection in the Treatment of General Paralysis', J Ment Sci, 1924, 70, pp 76 - 81. Claybury asylum reported remission rates of 33.5% in treated patients; but 60.5% in patients with short histories of the disease.

[115] See letter from Wadsworth to Board of Control, 16 May 1932: Wadsworth, superintendent of the Lancaster Asylum, asked the Board whether he could treat an uncertified patient at home with the permission and promised care of relatives, and observing the proper precautions. The Commissioners' reply was blasé; there was no reason why not if the patient had no objection: PRO, Kew: MH 51/698. See also G W B James, 'The Treatment of GPI', Lancet, Sept 3, 1927, pp 515-6: James discusses the possibility of treating at home, approving it as long as the patient's family is warned of the risks.

of patients: some of the earliest hospitals to report this were the West End Hospital for Nervous Diseases, the National Hospital Queen Square, and the Edinburgh Royal Infirmary. Each described patients diagnosed in out-patient departments on the basis of early clinical signs and Wassermann tests, and treated voluntarily on general wards. Variable degrees of improvement were noted, but results were generally claimed to be better than in asylum-treated cases.[116] The number of patients so treated rose through the 1930's; however, there was almost complete ignorance of the numbers involved and the comparative outcomes, and the Board of Control made no attempt to include them in official reports or studies.[117]

The most obvious location for early treatment before 1930 was the Maudsley Hospital, which had received unique powers in 1915 to admit voluntary patients, and was the flagship institution for preventive and out-patient psychiatry from the 1920's. During its early years malarial treatment was regarded by the Maudsley directors as experimental, and so more usefully employed in large mental hospitals where results would be more valuable.[118] As a result it was not used extensively, and the small number of general paralytics admitted from 1923 were instead usually treated with the alternative drug therapy tryparsamide, developed at the Rockerfeller Institute, and

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[116] See C Worster-Drought and H C Beccle, 'The Treatment of General Paralysis of the Insane by Malarial Infection', BMJ, Dec 29, 1923, pp 1256-7; 'Malarial Treatment of General Paralysis of the Insane', Lancet, Nov 8, 1924, p 959; Yorke, 1926, p 326; R Lees, 'Treatment of General Paralysis of the Insane by Induced Malaria', BMJ, Aug 22, 1931, pp 336-9.

[117] Meagher conceded that many more general paralytics might be under treatment in non-asylum institutions than the Commissioners realised, and that they might be milder than asylum cases: see Meagher 1929. See also 'General Paralysis ...', 1929, p 820: '[Malaria] was being used in general hospitals over and over again ... It was a matter of satisfaction that in this country more beds were being devoted to psychiatric work in the general hospitals themselves ...'; W D Nicol, 'The relation of syphilis to mental disorder and the treatment of GPI by malaria', Brit J Ven Dis, 9, 1933, pp 219 - 229. Col James was reported to have files of some such patients treated in general hospitals, but I can find no record of them - see Memo by Bond, 1 April 1935. PRO, Kew: MH 51/698.

unavailable to most asylums. As malaria therapy became established an increasing - although still limited - number of voluntary neurosyphilitic patients were treated, overseen by a medical officer who was responsible for their follow-up.[119] The Superintendent Edward Mapother concluded in the hospital's 1927 - 31 report that results in these patients were far superior to those obtained in other institutions, and were a good advertisement for early treatment: 'The difference merely shows what results might be obtained if only treatment in all cases were early; the moral is the importance of clinics such as the Maudsley to which patients will come at an earlier date than that at which they or their relatives in many cases can be persuaded by the family doctor that admission to a mental hospital is desirable. Many of these 'general paralytics' who have returned to employment are engaged in skilled and even responsible employment including one who was chauffeur to a member of the Cabinet in the last Government'.[120]

But despite such initiatives, by the end of the 1920's provision for early malaria treatment was regarded as far from adequate. Meagher noted in his report: 'The diagnosis of [GPI] in its early stages requires expert medical knowledge and laboratory resources. Our general hospitals provide these requirements, but few of them undertake the special treatment required, and nearly all of them decline to receive mental cases as in-patients. The psychiatric and neurologic clinics abroad,

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[118] See Report of the Maudsley Hospital for the year ending 31 Jan 1925, p 4.
[119] The total number of GPI patients received at the Maudsley between 1923 and 1931 was 360 (still only about 2% of the total number of patients treated over this period - a proportion remaining steady over the years); 207 of these were treated as in-patients and 153 as out-patients. It is not clear exactly how many of these received malaria therapy, but it appears to have been at least a half; see Annual Reports of the Maudsley Hospital (1923 to 1931).
[120] Report of the Maudsley Hospital for the years 1927-31, p 20.

with their easier conditions of admission, thus appear to possess advantages well worth considering.'[121] Disappointing results compared with those in Austria were commonly blamed upon rigorous British certification procedures: Wagner-Jauregg had always encouraged the treatment of early disease; and prophylactic malaria therapy based in Austrian syphilis clinics seems to have been commonly carried out.[122] In America too - at sites such as the Boston Psychopathic hospital - such work was attracting far more practical interest than in Britain.[123] The Horton hospital - rather than the Maudsley - was regarded as being the most successful at treating as early as possible; but was as restricted as other asylums before 1930 by the need to certify. One solution for contemporary doctors might have been a stretching of the bounds of 'insanity', which would allow certification for treatment earlier than strictly necessary. However, although this is difficult to judge, there is no clear evidence from clinical records that patients admitted to the Horton were less strictly 'certifiable' than would have been the previous norm.

Increasingly urgently, discussion of malaria therapy addressed the more general need for provision of early treatment: only such measures, it was argued, could prevent the accumulation of '... mental and physically bed-ridden wrecks which ... present the saddest picture of mankind's mortality.'[124] The 1930 Mental Treatment Act did finally make such provision by conferring optional powers upon

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[121] Meagher, 1929, quoted in E J Fitzgerald, 'Syphilis and "The Mental Treatment Act"', Brit J Ven Dis, 1934, 10, pp 117 - 137; on p 124.

[122] See Nicol and Hutton, 1937, p 172: 'In Vienna, patients are given malaria in general hospitals if found to have a positive CSF ...'

[123] For accounts of the Boston Psychopathic Hospital, see Lunbeck, 1994; I Skottoe, 'On the Methods in Vogue at the Boston Psychopathic Hospital', J Ment Sci, 1928, 74, pp 474 - 487. In both, malaria

local authorities to authorise voluntary treatment in mental hospitals and out-patient centres. The Act was the result of many years of discussion about the desirability of voluntary and early treatment of mental disease; and, perhaps surprisingly, there is no clear evidence that the promise of malaria therapy played a prominent part in stimulating the legislation. Nonetheless, the opportunities that it offered for this particular treatment were greeted optimistically. Fitzgerald, Assistant Medical Officer of Lancashire County Mental Hospital, noted with satisfaction that 'no longer must the incipient general paralytic be sent to a poor law institution to await the development of a certifiable disorder of the mind before he can receive malarial therapy. He may enter a mental hospital as a voluntary patient for the treatment of mental illness.'[125] Both Nicol the psychiatrist and Harrison the venereologist were fervent exponents of early treatment - and this included the treatment of those with a positive spinal Wassermann test and nothing more - potential or 'latent' neurosyphilitics. 'Malaria therapy' wrote Harrison, 'should be used as prophylaxis at the outset ... If, as I hope may eventually come to pass, we could make this form of treatment so convenient that any practitioner with a patient in the pre-paretic stage could send him for it with no more trouble than is caused by telephoning or writing a note, we should very largely wipe out the 1200 or so deaths per annum from GPI ...'[126] Curtis, Chief Officer of the LCC Mental Hospitals Department, also advocated

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therapy for voluntary patients is mentioned as a routine activity of the hospital, but not discussed in any detail.

[124] Meagher 1929, p 25.

[125] Fitzgerald 1934, p 124.

[126] L W Harrison, The Diagnosis and Treatment of Veneral Diseases in General Practice, 4e (London: OUP, 1931), p 422; see also Nicol and Hutton, 1937, who recommend malaria treatment of those with positive spinal Wassermann tests.

treating those with a very broad definition of neurosyphilis: '... If serological findings indicate that a condition exists which has not yet produced actual disturbance of conduct ... there is very good ground for giving the widest benefit of any doubt ... to the patient who has plenty of intelligent insight and is readily persuaded of his danger, and to ask for admission to a mental hospital [for malaria therapy] ... This may serve to illustrate the need, sometimes, for a generous interpretation of what is mental illness ...'[127]

In certain areas, such rhetoric was indeed reflected in practical exploitation of the Mental Treatment Act. Horton hospital was keen to establish itself as a preliminary treatment centre, and during the late 1930's increasingly advertised its facilities for voluntary patients and its dedication to preventive therapy.[128] Examination of its clinical case-books for this period does not give clear evidence of the proportion of voluntary patients treated, since records rarely noted whether or not a patient had been certified. However, one can chart the increasing number of patients admitted who were noted to have only very early mental signs, or no mental signs at all. Such 'latent' general paralytics included those whose CSF had been routinely tested because of a syphilitic spouse, or because of the birth of an affected child; a larger number had physical but no mental signs of GPI; some had tabes dorsalis which, although not itself regarded as amenable to malaria therapy, was

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[127] R H Curtis, 'Some Developments, Legal and Administrative, in Mental Treatment', J Ment Sci, 1938, 84, pp 183 - 202; on p 192.

[128] See Extract from Public Assistance Journal, 28 Jan, 1938. PRO, Kew: MH 51/698. The author notes that Horton hopes to 'encourage persons who are suffering from neurosyphilis to submit themselves for treatment as voluntary patients at the earliest possible stage ...'. In 1937 the Horton advertised its services in the BMJ, offering accommodation for private, voluntary and certified patients:

treated by some as potential GPI. Some patients had minimal mental symptoms, but were regarded as capable of understanding their illness and appreciating the need for treatment. Most were described as rational, having excellent insight, appreciating the nature of their disease, and willingly consenting to therapeutic malaria. Sources of referral reflected the eclectic nature of their disease: VD clinics, eye hospitals, neurological hospitals, and general hospitals as well as private practitioners. Patients who, according to my own assessment, fitted such 'voluntary' patterns did not appear in the Horton notes until 1937, when about 5% of admissions could be so classed. This figure steadily rose, reaching a peak in 1943 when about 21% could be classed as voluntary. Official publications put the figures much higher: by 1946, the hospital staff described the annual intake as: '... A small proportion of certified general paralytics and taboparetics ... a much larger proportion of relatively early cases who don't need certification ... [and] asymptomatic cases without any clinical features, but positive CSF on routine testing.'[129]

In tandem with early treatment, clinical descriptions during the period give a strong sense of the increasing autonomy of many neurosyphilitics. Patients now often appeared to be willing participants in their management; and although it is extremely difficult to assess the relationship of individuals to their therapy, their own views increasingly emerge. One Horton patient with early taboparesis, for example, told his

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see 'A Malarial Treatment Centre: The Unit at Horton Mental Hospital', BMJ, May 22, 1937, pp 1081-2.

[129] M Whelan and M H Bree, 'Conducing to the Cure: Social Psychiatry in the Treatment of Neurosyphilis by Induced Malaria', Lancet, Oct 5, 1946, pp 477 - 480; on p 477. My own estimates were made upon the basis of patients described as mentally normal or appreciating the nature of their disease. The discrepancy with contemporary statements demonstrates how difficult it is to make

doctor that malaria treatment had greatly improved his condition in the past, and requested a repeat course. Another, anxious that his symptoms of GPI might return, insisted upon a repeat of his therapy of two years before.[130] Braslow discusses malaria therapy in the context of 1930's California state hospitals, when there was a sharp upswing in the number of voluntary admissions amongst neurosyphilitics - many of whom were asymptomatic except for positive Wassermann tests.[131] He argues that malaria therapy (pace the anti-psychiatrist reading) allowed patients to become active participants in their treatment regime, fostered empathy between doctors and patients, and transformed the image of the asylum from that of institution of confinement to that of place of cure. The Horton notes certainly confirm this image of greater patient participation, and of a degree of negotiation between doctor and patient. How far this was peculiar to malaria therapy, and how far to voluntary psychiatry as a whole, is a question which would need further research to elucidate.

The Maudsley too continued to treat voluntary neurosyphilitics through the 1930's and 1940's, although not in markedly increased numbers.[132] Between 1932 and 1935 there were proposals to set up a Continental-style neuropsychiatric clinic on site to study and treat patients with incipient organic mental disease. Early prevention and treatment of neurosyphilis through co-operation with VD clinics, general hospitals and asylums were to be a central project of this centre. A combination of

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retrospective classifications; one possible conclusion is that a number of patients not apparently 'sound of mind' were nonetheless received as voluntary patients.

[130] Clinical case-books of Horton Hospital for 1943.

[131] See Braslow, 1995.

[132] Figures are available only until 1935: from 1932 to 1935 176 GPI patients were treated (1.5% of total numbers treated); again it is not clear how many received malaria therapy. See Annual Reports of the Maudsley Hospital (1925 to 1935).

wrangles over the building of the clinic and the arrival of the war, however, prevented the plans from materialising.

Despite changes at the Horton, however, the implementation of early treatment after the Act was - surprisingly - regarded as inadequate and disappointing. By the post-war years, there was widespread feeling that preventive implementation of malaria therapy had not been achieved: only three British papers had been written on the subject, and nowhere had it been extensively used outside the Horton itself.[133] Harrison and James reportedly made strenuous efforts during the early 1930's to establish a centre purely devoted to prophylactic and early malaria therapy; but claimed that they found little enthusiasm amongst colleagues and hospital authorities.[134] This perceived failure was partly related to perceptions of the Mental Treatment Act as a whole. It was commonplace until the beginning of the war to complain that voluntary treatment was being taken up slowly and inadequately; this was blamed, amongst other factors, upon organisational difficulties, pressure of beds, the resistance of individual psychiatrists, and the clinging stigma of mental disease.

But there were also difficulties more specific to neurosyphilis, and these echoed the pragmatic problems which had affected the implementation of the Wassermann test itself for at least twenty years. One concerned the wide variety of

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[133] See E L Hutton, 'Recent Advances in the Aetiology and Treatment of Neurosyphilis', J Ment Sci, 1941, 87, pp 1 - 49; on p 40: 'It is highly regrettable that prophylaxis has been so little adopted in this country, though it has been adovcated and employed on the Continent for the last ten years or more.' See also Lescher and Richards, 1944.

[134] S P James, 'Remarks Preceding a Demonstration Relating to the Practice of Malaria Therapy at Horton', Brit J Ven Dis, 1933, 9, pp 230-4; on p 230: '... He wished it were possible to stimulate more people to voice a demand for malaria therapy under conditions in which it could be given with the greatest possible success ... perhaps [then] ... the authorities would move a little more quickly in the matter ...'; Harrison later reported: 'For years I have tried to get arrangements made that malarial treatment may be easier to obtain than it is at present ...', quoted in Nicol and Hutton, 1937, p 168.

medical services dealing with the disease. Communication and co-operation between VD clinics, general hospitals, mental hospitals and psychiatric clinics was vital to successful early treatment of neurosyphilitics. The Act did not, however, seem to strengthen this 'weak link in the preventive health service of the nation'; and throughout the 1930's and 1940's there were continual calls for better liaison between venereologists, neurologists and psychiatrists.[135] Again, whilst widespread CSF testing of syphilitics was invariably recommended, it was only patchily carried out.[136] Wariness of the lumbar puncture continued to pose a serious obstacle: 'If it could happen', wrote Harrison, 'that all persons with a history of syphilis could have their spinal fluid examined at intervals, or even if the medical profession had learned to have the fluid examined whenever a patient showed the least signs of central nervous disease, I think we could save many a household from the misery, the financial loss and ... the shame, which general paralysis brings.'[137] But lumbar punctures were still not regarded as trivial procedures: doctors themselves were often nervous of them; and patients were discouraged from submitting to the test by the prospect of possible prostration for several days afterwards. 'There are still many physicians ...', wrote Hutton, 'who regard a lumbar puncture as a minor surgical operation ... In view of this the patient is inclined to regard it as a major operation and is unwilling to submit to so drastic a procedure, especially when feeling well ... The publicity necessarily

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[135] Fitzgerald 1934, pp 131 and 135.
[136] See Nicol and Hutton 1937, p 164: '... The present unsatisfactory recovery rate in GPI cannot be appreciably improved until it is recognised in this country that examination of the CSF is as essential as examination of the serum ...'
[137] Quoted in Fitzgerald, 1934, p 121.

connected with even a brief stay in hospital naturally engender an even greater reluctance.'[138]

On a more theoretical level, the question of asymptomatic neurosyphilis was still a thorny issue. Many doctors pointed out that there was no certainty that all such cases would progress to GPI; and that the risk of therapy was unjustified in apparently healthy patients. There was disagreement as to whether such early neurosyphilitics should be treated with malaria or simply with arsenicals, such as the promising tryparsamide. Indeed many doctors preferred to concentrate their efforts upon the prevention and early treatment of syphilis per se - arguably a more achievable goal than the treatment of early neurosyphilis. The views of patients increasingly entered these arguments. People healthy apart from a positive laboratory test, it was pointed out, could not easily be persuaded to submit to the risk, inconvenience, or stigma of fever therapy. The need to educate and encourage patients to receive early treatment was acknowledged, but it was clearly not an easy task.[139]

After the Asylum: General Paralytics Enter the Community

By the early 1930's, enough time had elapsed since the introduction of malaria therapy for interest to turn to the long-term fate of those released from asylums. Psychiatrists had never underplayed the desirability of following up discharged patients to assess long-term results - although the practical arrangements had proved difficult. Meagher's 1929 study was the first large-scale attempt to study outcomes in

[138] Hutton 1941, p 42.

ex-asylum patients, using information from doctors, letters, and personal visits to families. As I have suggested, one aspect of his report which was played down by commentators was the disheartening image of patients in the community with varying degrees of mental deficiency. Whilst Meagher extolled the practical benefits of malaria treatment, he also hinted clearly at the limits of these benefits and their cost to patients and families - and he noted that the longer the period of time studied, the poorer the long-term results of patients were. He also acknowledged the objection that hospitals using malaria therapy might acquire an enthusiastic 'urge' to discharge general paralytic patients during a period of remission, who were not really suitable for life in the community.

After Meagher, an increasing number of studies concentrated upon the fates of discharged general paralytics, and results confirmed that only a proportion - ranging from a tenth to a half - were maintaining their recovery.[140] Studies such as these, however, dealt in figures, not descriptions, and did not give the impression of widespread mental deficiency or social difficulties which Meagher had drawn attention to. The Horton clinical records demonstrate the range of conditions which could be covered by bald categories such as 'discharged recovered'. The proportion of treated patients who were discharged varied over the years - but the average figure between 1925 and 1937 was 40%.[141] From soon after the opening of the centre, there were attempts to follow up every patient after discharge; work carried out by a social-

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[139] All of these points were raised in a discussion of Nicol's paper: W D Nicol, 'The Relation of Syphilis to Mental Disorder and the Treatment of GPI by Malaria', Brit J Ven Dis, 1933, 9, pp 219 - 244.

[140] See, for example, W Reid, 'General Paralysis: Results of Eight Years' Malarial Treatment', J Ment Sci, 1932, 71, p 67; 'Malaria therapy for GPI', BMJ, Aug 1, 1936, p 234.

worker who sent contact letters and attempted to visit patients in their homes. The earliest treated patients had their first documented contacts during the early 1930's - five or six years after their discharge - when the phrase 'Miss Dale called', or 'Miss Bree visited' became a familiar part of clinical records.

These records offer only a rough guide to the outcome of patients, and are very difficult to interpret retrospectively. However, approximately a half of all discharged patients were noted to have had a satisfactory outcome - in the sense of remaining stable for many years of their remaining life. At best some of these were described as well, earning a living, maintaining themselves and their families, or 'in excellent health and normal in every way'. The social-worker would often emphasise, in support of this, the cleanliness of the home or the well-being of the family. However, many in this category were evidently mentally dependent: they might report suspicions of being followed; or a family member might note that 'something was not quite right about her nerves', or that 'he was peculiar'. Comments such as these echoed Meagher's descriptions, and we can guess that the number of patients claimed as truly maintaining a recovery was optimistic. The remaining half of discharged patients fared even worse: some were readmitted to an asylum after a short time; some were untraceable; some were reported to have become criminals, vagrants, alcoholics, or to have committed suicide in the intervening years. A note of caution about the mental state of patients following treatment increasingly entered doctors' discussions. Dr Golla, director of the London Central Pathological laboratory for much of this period, received numerous patients discharged from the Horton in order

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[141] Clinical case-books of Horton Hospital (1925 to 1945).

to perform follow-up tests. He remarked: 'They had people who went back to their skilled duties - civil servants, actors, etc - but he thought that of few could it be said that they were the people they had been before the onset of their mental illness ...'[142]

But the development of the social work profession ensured that at least some of these discharged and dependent neurosyphilitics were not being ignored. During the 1920's after-care - although increasingly discussed - had still been predominantly the remit of philanthropic organisations such as the London After Care Association. Meagher's report had emphasised the need for after-care of discharged patients, and had noted that this charity was becoming involved in such cases in London.[143] Such possibilities, however, were evidently limited, and it is unlikely that most provincial asylums were able to follow up patients satisfactorily - whether through home visits or through clinical assessment and Wassermann testing. After 1930 the passing of the Mental Treatment Act and the establishment of the Association of Psychiatric Social Workers set the scene for a slowly increasing involvement of social workers in psychiatric practice. Whilst they struggled to secure their professional status, the Horton hospital was quick to incorporate such input into its own practice.[144] Here, social workers developed work with neurosyphilitics into a small sub-specialty during and after the war. Marjorie Bree was most involved with the Horton during this period, and published a number of articles based upon her work there. The social worker's functions were strongly reminiscent of those employed at the Boston

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[142] Quoted in Nicol and Hutton, 1935, p 816.

[143] The records of the After-Care Association, held in the Wellcome Institute CAMC collection, make no specific mention of care of discharged neurosyphilitics; but diagnoses are generally not specified anyway.

Psychopathic Hospital before the introduction of malaria therapy.[145] They included taking detailed histories of social background and syphilis infection; tracing contacts to encourage testing; explaining the disease and treatment to relatives - including the risk of malaria relapse after discharge; offering psychological support to families, and helping practically with finances and employment - for example persuading potential employers of the need for 'some necessary special consideration and a disregard of minor peculiarities'.[146] Social workers were keen to make their own contribution to education, emphasising that they regarded neurosyphilis as a medico-social problem, 'relieving those concerned of the unwarrantable and exaggerated fears which the word syphilis conjures up in the ignorant...'[147] The Horton also had a well-organised system for clinical follow-up assessment and post-discharge Wassermann testing, organised by a special medical officer and based at the hospital itself and the Maudsley clinic. Social workers played a role here too - explaining to patients the need for continued testing, followed up absentees, attending the clinics to interview and advise relatives, and reporting on social adjustment.

Until well after the war such facilities were limited both in London and in the provinces. Co-operation between asylums and VD clinics continued to present problems; and the Board of Control's 1924 instructions that the medical officer of health should be informed of neurosyphilitic discharges to enable follow-up fell into

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[144] For a general history of psychiatric social work, see N Timms, Psychiatric Social Work in Great Britain (1939 - 1962) (London: Routledge and Kegan Paul, 1964).
[145] Lunbeck, 1994.
[146] Quoted in the Handbook on Mental Health Social Work (London: LCC, 1947), p 72.
[147] Ibid, p 73; for further aspects of social work in the context of the Horton see M H Bree, 'The Function and Use of Relationship between Client and Psychiatric Social Worker', Br J Psych S W, June 1952, 6, pp 27 - 32.

abeyance in many areas.[148] Horton managed to offer after-care to 228 of 1200 cases treated between 1942 and 1952; but the proportion at other hospitals is likely to have been much smaller.

The burden of discharged general paralytics in the community was not the only legacy of malaria therapy. Equally frequently discussed were those whose treatment had arrested the progress of their disease; instead of dying rapidly as formerly, they were accumulating in asylums as chronic mental patients. Psychiatrists had pointed out soon after the introduction of the treatment that it might not be entirely desirable if it arrested the course of GPI without producing improvement sufficient for discharge. As Yorke noted in 1926: 'In many cases patients ... become clean in their habits but can't return to work ... From the purely utilitarian point of view this can only be described as a disastrous result ... But', he continued wryly, ' ... if prolongation of life is counted good, then it must be good ...'[149] Such a problem only emphasised the need to treat earlier rather than later: Riddoch echoed the warning: 'Personally, I consider that [malaria treatment] ought to be restricted to early cases, for no useful purpose is served in bringing back to life patients so deteriorated that improvement would be insufficient to allow of their discharge from a mental hospital.'[150]

Ten years later, Nicol wrote that a large residue of arrested general paralytics was indeed accumulating in mental hospitals ...'[151] By the 1940's this population of chronic inmates, resulting from dubiously successful treatment, was attracting some

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[148] See Fitzgerald, 1934, p 122.
[149] Yorke 1926, p 427.
[150] G Riddoch, 'Neurosyphilis', Brit J Ven Dis, Jan 1928, 4, 1, pp 1 - 13; on p 6.

despair and frustration. The assessment of how serious this problem was differed between observers. In a post-war article entitled 'The Chronic Paretic', the ex-Freirn psychiatrist Kirman reminded readers of Meagher's estimate twenty years previously that a third of treated general paralytics would become chronic asylum inmates. He described the common perception that there were now large numbers of 'defect-cured or stationary paralytics who fill our asylums in great numbers, and are a burden to their families and society.' Nonetheless, he noted a consolation; that such patients were often fixated at the hypomanic stage, and as such had an '... almost pathological appetite for manual work, such as scrubbing, sweeping, cleaning and scouring. They were expansive, cheerful, indefatigable workers who would gladly work an extraordinary number of hours without reward.' The accumulation of chronic schizophrenics was, he concluded, a far more serious problem; only a small proportion of asylum accommodation in England was taken up by paretics, 'and some of them appreciably more than earn their keep.'[152] Nonetheless, Nicol replied to this article with an admonishment that the problem of the chronic paretic could not be ignored: 'The number [of paretics] in our mental hospitals will never be adequately reduced until the diagnosis is made in the earliest, and preferably the latent, stage of the disease, and appropriate treatment is immediately initiated, either with the hope of cure or as a prophylactic measure against the subsequent development of an incapacitating form of mental disorder.'[153]

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[151] Nicol and Hutton, 1935, p 815.
[152] B H Kirman, 'The Chronic Paretic', Lancet, Nov 22, 1947, pp 755-7.
[153] Letter from W D Nicol, 'The Chronic Paretic', Lancet, Dec 13, 1947, p 891.

Despite these problems, the number of patients diagnosed with neurosyphilis had been falling steadily since the second decade of the twentieth century, and the use of malaria therapy too gradually declined.[154] Its end was speeded by the advent of penicillin, introduced into Britain during 1942, and soon used in all stages of syphilis. At first penicillin was used only as an adjunct to malaria therapy; but by the 1950's it was thought to be just as effective alone, and to be far safer besides.[155] After 1955 there were few references to malaria therapy in the medical literature, although it continued to be used occasionally in resistant cases until the 1960's.

But as GPI disappeared from the public eye there were reminders that the legacy of malaria therapy remained. The discharged neurosyphilitic continued to be of interest to the social worker - particularly as community care continued to gain interest in the field of mental health. In 1960 Bree published a report on the 'dement in the community', drawing upon her long experience of treated neurosyphilitics. Of 1200 cases treated at the Horton between 1942 and 1952, she concluded, 11% had made a complete recovery; the rest were now demented. Most of these were cared for by their families, '... maintained with commendable affection and loyalty and only very rarely with a sense of martyrdom.' She noted also, however, the frequently adverse effects upon the supporting family, echoing the concerns of Meagher thirty years previously: 'Community care is a double-edged weapon, and it is sometimes hard to be certain that we may not be doing more harm than good.'[156]

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[154] The reason for this decline was much discussed - a subject I shall return to in the following chapter.
[155] See Leader: 'Treatment of Neurosyphilis', BMJ, Oct 21, 1950, pp 934-5.
[156] M Bree, 'The Discharged Dement', Lancet, July 23, 1960, p 217.

The Impact of Malaria Therapy

In each of the previous chapters, episodes in the history of GPI have demonstrated both uses of the disease for the psychiatry profession, and gaps between rhetorical claims and practical advances. Malaria therapy, too, can be interpreted in this light. Triumphalist accounts were common in the years after its introduction, and have persisted in historical writings to the present day. Its undoubted practical effects upon the hitherto most final of mental diseases made it a strong advertisement for the efficacy of modern psychiatric treatment; and as such it was actively promoted by the Board of Control in the face of perceived reluctance amongst asylum doctors. In many senses it can be regarded as the first of the heroic physical treatments of the twentieth century; although such a concept was not explicitly discussed until during and after the Second World War. From the 1940's fervent exponents of these treatments such as William Sargant battled with critics who championed psychotherapeutic methods, and who increasingly condemned physical methods as the new form of restraint.[157] Sargant characterised the future as belonging to those who practised bold, eclectic therapies which gave results - however theoretically obscure or emotionally repellent: 'It is into the hands of the bold experimenters', he proclaimed, 'that success has fallen.'[158]

In this climate, malaria therapy did indeed appear to have sounded the keynote; it was often hailed as the breakthrough which had marked the beginning of

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[157] See, for example, W Sargant, 'Aim and Method in Treatment: Twenty Years of British and American Psychiatry', J Ment Sci, 1954, 103, pp 699 - 709.
[158] Sargant and Slater, 1946, p 8.

the modern era in psychiatry. 'The introduction of malarial treatment in Vienna in 1917', Sargant wrote, 'opened a new chapter in psychiatry, of which we have as yet in this book turned only the first page or two. We cannot foresee what the future may bring; but we can be sure that the field of organic treatment, in which Wagner-Jauregg scored the first brilliant successes, will still be expanding for generations to come ...'[159] Rees' Presidential Address to the MPA made the same point: 'The modern era in psychiatry with its emphasis on active treatment may be said to date from the introduction of malaria therapy ... The elimination of general paralysis has transformed our infirmary wards, which thirty years ago were regarded as the most active in the hospital.'[160]

Despite this enthusiasm, few claimed that malaria had eliminated GPI. The decline of the disorder - which I discuss in the following chapter - was rarely attributed in any part to the new treatment; although some suggested that it might have reduced the number of deaths returned as 'GPI' by allowing patients to die of intercurrent diseases. To psychiatrists the tangible effects were those of greater manageability of patients, and the movement of patients from the care of the asylum to the care of the community. It was such pragmatic benefits which doctors concentrated upon, despite the patent confusion in defining and assessing results - and the lack of rigour even in the context of contemporary experimental design. The ability to discharge was a visible indicator of the power of the new therapeutics; it fitted well into the developing ideal of psychiatry in the community (incidentally

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[159] Ibid, p 148.
[160] T P Rees, Presidential address: 'Back to Moral Treatment and Community Care', J Ment Sci, 1957, pp 303 - 313; on p 308.

inspiring a well-developed social work service at the Horton); and it relieved pressure upon overstretched institutions.[161] Sargant constantly referred to this benefit as he contrasted the old days of asylum back-wards filled with chronic patients, with the present situation in which new treatments allowed the rapid turnover of patients. Although the process of malaria therapy was probably gruelling even for nurses, the overall effect of discharges upon the morale of asylum staff was probably considerable: 'I well remember', wrote the Bexley psychiatrist Cooke, 'the enthusiasm engendered in the nursing and medical staff by malarial treatment, which created a spirit of cheerful activity in wards oppressed by the hopelessness of such [GPI] cases.'[162]

Such were the benefits of malaria therapy for psychiatrists; but in several ways its implementation created difficulties and disappointments. Deaths and complications continued to cause unease; and it is possible that ordinary doctors were more concerned about the treatment's safety than is immediately obvious. This was certainly the perception of the Board of Control, and it may have been a significant reason for the less widespread implementation of the treatment than on the continent. On the other hand, there is no evidence of any public censure of the malaria therapy; and the stance of the Board, if not of every doctor, largely bears out accounts of ethical attitudes before the Second World War: 'The history of medicine reveals again and again the bias that exists towards proving the triumph of benefit over risk - and at

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[161] Fennell refers to the last point in the context of psychosurgery: Fennell, 1996, p 142.
[162] L C Cook, 'The Place of Physical Treatments in Psychiatry', J Ment Sci, 1958, pp 933 - 942; on p 936. See also Rees, 1957.

times this has involved extraordinary denial of some unpalatable truths.'[163] By the time the Nuremberg code paved the way for modern conceptions of medical ethics, malaria therapy was no longer of major relevance - although it is possible that the rationale of contemporary psychiatrists would actually have satisfied this code.[164] A comparison with the United States would be interesting: here, psychiatrists were reputed to have received the therapy particularly enthusiastically during the 1920's, and to have exploited voluntary and early treatment far more widely - but to have experienced a far more vigorous ethical backlash.[165] Partly because of the perceived dangers of malaria, they exploited 'artificial' forms of fever therapy, such as electropyrexia and the Kettering hypertherm, to a far greater extent than British psychiatrists[166]

Whilst the treatment allowed the conspicuous discharge of general paralytics from asylums, it paradoxically also created new pressures through an accumulation of chronic 'arrested' neurosyphilitics - both within asylums and the community. Reports from Europe during the 1940's and 1950's suggested that this build-up of neurosyphilitics was posing particularly severe problems, and their plight only served

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[163] C Medawar, Power and Dependence: Social Audit on the Safety of Medicines (Social Audit Ltd: London, 1992), pp 12 - 13. Fennell characterises this stance as typical of the 'age of experimentation' in psychiatry: Fennell, 1996, p 150. For an account of the development of ethics in US medicine, see D J Rothman, Strangers at the Bedside (New York: BasicBooks, 1991); and (a shorter account) L L Booth, 'Clinical Research' in Bynum and Porter, 1993, Vol 1, pp 205-29.

[164] This 1958 comment could equally have applied to an earlier doctor using malaria: 'Nobody likes the physical treatments in psychiatry: they are cumbersome, often lengthy, and have their dangers, but when selected and administered with thoughtfulness and care, their failures and occasional disasters are infinitesimal compared with the benefits they bring ...' Cook, 1958, p 941.

[165] Canadian psychiatrists, in contrast, began to use the treatment only in the late 1920's.

[166] See, for example, the admiring report of A J King, 'Fever Therapy at Dayton, Ohio', Brit J Ven Dis, 1937, 13, pp 267 - 274; and E Pegg , 'The Nursing Aspect of Hyperthermy Treatment', Brit J Ven Dis, 1943, 19, pp 166-9. Both of these reports give unusually detailed accounts of patient reactions to these treatments. Such treatments were advocated to a limited extent in Britain; see for example N B

to re-emphasis that general paralysis was still a disabling and inexorable degenerative disease. For this reason concerns were increasingly expressed about the long-term benefits; particularly since - with the exception of the Horton social work service - facilities for supporting families were probably very inadequate. The Horton itself was the most conspicuous success resulting from malaria therapy; and it was here that highest success rates and lowest death rates were claimed. But the greatest benefits were reaped by tropical medicine: research into malaria itself attracted far greater interest and investment, largely for imperial reasons which were accentuated by the arrival of the war. It was this work, too, that would be built upon in the future - whilst malaria therapy yielded few new insights into psychiatric disease, and neurosyphilis itself would soon be a disease of the past.

It was at the Horton that malaria therapy allowed doctors to take most advantage of the new climate of voluntary treatment - to a far greater extent than the Wassermann test alone. For a variety of practical reasons, however, early treatment of neurosyphilis never reached its full potential; and by the time there was a more general upswing in voluntary treatment during the 1950's, GPI was a declining disease and the opportunity had been largely missed. The advent of penicillin and the decline of neurosyphilis finally ended the experiment of malaria therapy; and it must be remembered that, despite the high profile of the disease, it affected only a small - and decreasing - proportion of psychiatric patients. In this sense the treatment appeared to have arrived too late to be fully explored; and it is possible that popularly

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Graham, 'Some Remarks on the Treatment of General Paralysis by Diathermy', J Ment Sci, 1933, 79, pp 89 - 93.

recognised initiatives such as the general campaign against syphilis to a certain degree stole its thunder.

For the first time, however, the effects of this chapter in the history of GPI can be appreciated from the point of view of the general paralytic. The majority of published and hospital sources, it is true, keep the usual author - the doctor - firmly in the foreground, with the patient himself the shadowy object of treatment. Protocols, nursing requirements, complications, and results are described objectively; with anecdotal details of individual case-histories occasionally offering a more personal and colourful view of the patient's predicament, or his attitude towards his treatment. The therapy, nevertheless, was becoming popularised amongst the general public in a limited way during the 1920's. Newspaper reports heralded the new hope for sufferers of mental disease; and members of the Board of Control noted that patients and their friends were already requesting and enquiring about the treatment by 1923. As the 1930's progressed, the image of the patient moved steadily into the foreground, in parallel with the move beyond the asylum and an impression of the broadening of the patient's life-history. The pre-asylum life of GPI patients had always had a place in asylum clinical notes, as the disease was regarded as the culmination of past experiences, faults, and - latterly - venereal disease. More recently, this past had become even more of a continuum with the asylum present, as many patients had had earlier identities as 'syphilitics' or 'early neurosyphilitics' - treated at VD clinics and psychiatric out-patient departments before their admission. Now, with the possibility of discharge following malaria therapy, such patients were increasingly portrayed as

having a future beyond the asylum - their hospital stay being only a transient stage in their life history.

Fennell interprets the 1930 Mental Treatment Act in terms of social control - suggesting that, by allowing doctors to foster somatic therapies with extensive legal immunity, it created a climate in which the patient could be further exploited.[167] Braslow suggests, in contrast, that in the American context malaria therapy had an extremely positive impact upon the relationship between doctors and patients. Both accounts contain some truths in the context of British malaria therapy. Braslow's suggestion that the treatment led doctors to regard their patients in a more positive, approving light is not strongly evident from the Horton archives. Notes from the 1930's, for example, still contain a mixture of more or less morally charged comments - although none that necessarily relate specifically to censure against general paralytics: 'he lived a wild, gay life'; 'had loose morals'; 'appears a man of excellent moral character, but obviously knocked about a bit in his youth at sea'; 'faulty in habits'; 'silly and childish'.[168]

Nonetheless the stated intention of the social work department - to move syphilis from the sphere of the moral to that of the medical and social - was evident in other ways. From the 1920's the notes included increasingly detailed accounts of patients' families, personalities, and social circumstances. Dialogues between doctor and patient were increasingly recorded, including the wishes of the latter concerning their treatment. That malaria had a powerful effect upon the imagination of patients

[167] Fennell, 1996, pp 107 - 119.
[168] Clinical case-books of Horton Hospital.

is undeniable. A letter written by a Claybury patient to the Visiting Commissioners - ironically showing a degree of grandiosity - described mosquitoes as 'God's hypodermic syringes': 'I know', he enthused, 'that if any spirochaetes were lurking in my system malaria parasites would quickly destroy them ... Amazing results have been achieved everywhere ... Thousands of husbands have returned to wives ...'[169] More moderate letters to asylum doctors and the Board of Control give some idea of the initially positive impact that malaria therapy had upon individual lives. The wife of a recently discharged patient wrote: 'My husband has been much better mentally since the treatment, for which I am very grateful'; another patient himself wrote: 'I thank you very much for what was done for me, also thanks to the staff, during my sickness. I am pleased to let you know I have re-started work in my own trade.'[170] In his report Meagher emphasised 'the many spontaneous expressions of gratitude by both patients and relatives for the benefit they attributed to the special treatment received, and for the kindness and interest of the MOs of the hospitals where they had been treated ... Some of the recoveries were described as marvellous - as miracles ... malaria treatment has achieved for itself considerable popular esteem.'[171] Many families - particularly, perhaps, those who benefited from the Horton's enthusiastic social work department - may well have felt that malaria therapy had been worth-while. This aspect of treatment benefit, in the face of a hopeless and degrading disease, is one that tends to be glossed over in accounts such as Fennell's.

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[169] Letter by patient no. 7887, Claybury Asylum. PRO, Kew: MH51/698.
[170] Quoted in Annual Report of the Board of Control for the year 1931 (London: HMSO, 1932), p 128.
[171] Meagher 1929, p 52.

On the other hand, many families now faced the great burden of caring for dependent, difficult relatives with as yet inadequate after-care and support - relatives who twenty years earlier might have died quickly in the asylum. Letters expressing less happy reactions were not common (or were perhaps not kept), and extremely unhappy after-histories tended to be played down in reports: only those by Meagher and Bree give us an idea of the reservations of ex-patients and their families. So whilst malaria indeed seemed to have a good reputation, and whilst patients were requesting their own treatment more and more, the attitude of the average general paralytic towards the benefits, risks, and ordeals of his treatment remains largely unknown.[172] Increasing patient autonomy did not alter the truism that - with few constraints on practice and with the enthusiastic backing of the Board of Control - doctors had a largely captive audience for their first experiment in physical therapy.

[172] Neither, unfortunately, do there seem to be any available accounts by survivors of malaria therapy.

CHAPTER 5

THE RISE AND FALL of GPI

Introduction

The timespan during which GPI had an important place in psychiatric practice lasted about a hundred and twenty years. In Britain, the first two thirds of this period saw its alarming rise, which culminated just after the turn of the twentieth century as GPI appeared to epitomise the threat of racial degeneration. Thereafter - just before the disease stimulated the first of the major psychiatric 'physical' therapies - it abruptly declined, so that by the 1950's new cases were extremely rare. Although the reasons for this decline prompted a certain number of conflicting opinions at the time, it was soon agreed that preventive and therapeutic advances against primary syphilis were the main explanation. This account, of course was a success story for medicine as a whole; and psychiatrists themselves could take little specific credit despite their hopes for malaria therapy. It became accepted wisdom, too, for those later historians who turned their attention to GPI - although they rarely scrutinised the details of the epidemiological changes.

During the 1970's, revisionist historians began to challenge the traditionally accepted efficacy of medical advances, as they suggested that more general trends such as improved nutrition and population stability were far more important in ameliorating the major infectious diseases such as TB and smallpox. Several years before this, however, the psychiatrist Edward Hare challenged the standard account for GPI in service of a different thesis, by presenting a historical

epidemiology which attributed its rise and fall to the natural processes of infection spread. Hare's interpretation took it for granted that diseases such as GPI could be traced as objective entities through history, and could be interpreted in the light of modern understanding. Supporting as it did his stringently biological approach to mental diseases, his work can be regarded as the final chapter of a story in which GPI had been used, by successive generations of psychiatrists, to make claims for the medical status of their profession. My primary aim is not to support or refute Hare's story. Although I suggest that his is the most unified and satisfactory explanation of observed events, it remains a hypothesis clearly in service of a scientific theory - and beyond the remit of my own historical account. More interesting, perhaps, is the observation that Hare's work can be approached both as a current scientific or historical interpretation amenable to criticism, and as a final part of the history of GPI itself - a psychiatrist's response to the last decline of a vivid and important disease.

The Rise of GPI

The appearance of an apparently new disease category during the early nineteenth century was a puzzle to Victorian alienists. The majority, however, took it for granted that GPI had existed long before the 1800's, and that earlier doctors had simply neither recognised its symptoms nor forged them into a nosographical entity.[1] Hare, arguing that GPI was in fact a recent disease, explained the dominant view of Victorian physicians upon the basis of their belief that diseases

[1] See Austin, 1857, quoted in Hunter and Macalpine, 1963, p 1054: 'General paralysis, though it had doubtless existed from the earliest period of insanity ... never so fixed the attention of those who must have witessed it, as to be recognised and described as a distinct disease, till the early part of the present century.'

were enduring and immutable, and that GPI was caused by sexual excess and alcoholism - which could hardly be regarded as new vices.[2] Neither point can be entirely corroborated: as I shall describe, doctors were already interested in the mutability of disease well before GPI began to be documented; and mid-nineteenth-century views of GPI linked it predominantly to the rise of urban ills and capitalist ambition - both factors which would have fitted well with an emerging disease.[3] A more likely rationale was the triumphalist view of medical progress which tended to doubt the abilities of earlier observers and hail the advances of the new era in psychiatry. Generally British doctors were happy to praise the work of Frenchmen such as Bayle and Calmeil, as well as improvements in the organisation of asylums which allowed a greater recognition of GPI. However, an enthusiasm for making retrospective diagnoses from more ancient records - which enabled them to identify GPI in Shakespeare's plays - also allowed them to claim that British doctors such as Willis and Haslam had in fact been the first to describe the disease. In 1864, for example, Sankey, the Superintendent of Hanwell Asylum, asserted that ' ... there is sufficient evidence at once to settle the question that the disease is old, and that its diagnosis only is modern ...' He proceeded to quote a passage written by Thomas Willis in 1672 which, he claimed, carried back two hundred years the date of observation of the symptoms.[4] Mickle's later authoritative text on GPI gave a classic account of the

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[2] Hare, 1959, pp 605-6.

[3] See Austin, 1857, quoted in Hunter and Macalpine, 1963, pp 1054-5: 'Whether this increase [of GPI] is real, may perhaps be doubted ... The more easy recognition of the malady has possibly produced an apparent increase; though, on the other hand, it is equally likely that the wear and tear of modern society ... and the more frequent occurrence of mental anguish ... may have actually augmented the number of its victims.'

[4] W H O Sankey, 'The Pathology of General Paresis', J Ment Sci, 1864, 9, pp 467 - 493; on p 468. See also D Skae, 'Contributions to the Natural History of General Paralysis', Ed Med J, 1859-60, 5, pp 885 - 905; on p 885: 'Haslam ... refers to the symptoms of this disease in terms which show

journey from ignorance to enlightenment: '... The light of the first knowledge of the disease gleams in the pages of Willis (1672); ... Haslam (1798 - 1809) caught flashes and sparkles of the truth in his discerning appreciation of some of the chief features of the affection ... the discovery was completed by Bayle ... closely followed by Calmeil (1826) ...'[5] This remained the standard line, despite refinements in the explanation offered. In 1923, for example, George Robertson added that the Napoleonic Wars had concentrated a large number of military GPI cases in the major mental hospitals of Paris; this, combined with the ordered regime and careful documentation introduced into asylums by Pinel, allowed the symptom complex to be identified clearly for the first time.[6] Later twentieth-century historians, too, have predominantly followed this line.[7]

The question of the subsequent epidemiology of GPI was more difficult to pin down. Arguments raged throughout the nineteenth century as to the real meaning of an apparent rise in all cases of insanity: some claiming an actual increase in disease; others claiming accumulation in asylums, a change of public view leading to 'an ever-increasing tendency to remove the insane from the 'mass of unregistered lunacy to 'official cognisance'', and a broadening of the limits of certifiable insanity.[8] However this dilemma was resolved, asylum figures for the

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that he had recognized some of its distinctive features, but he failed to follow up his enquiries so as to make out its natural history ...'; and T Claye Shaw, 'On the Antiquity of General Paralysis', J Ment Sci, 1869, 14, pp 460-5.

[5] W J Mickle, General Paralysis of the Insane, 2e (London: H K Lewis, 1886), p 2.

[6] G M Robertson, 'The Discovery of General Paralysis', J Ment Sci, 1923, 69, p 1.

[7] See, for example, Rosen, 1968, p 250, who attributes GPI's long period of non-recognition to the fact that earlier observers were using superficial, symptomatic criteria to identify disease entities. Not until the clinico-pathological approach of the Paris school, he argues, could GPI be accurately delineated.

[8] Stewart, 1896, p 760. For a variety of solutions to this problem, see E Hare, 'Was Insanity on the Increase?', Brit J Psychiatry, 1983, 142, pp 439 - 455; A Scull, Social Order/ Mental Disorder (London: Routledge, 1989), esp Ch 9; M A Arieno, Victorian Lunatics: A Social Epidemiology of Mental Illness in Mid-Nineteenth-Century England (London and Toronto: Associated University Presses, 1989).

second half of the century seemed to suggest that the proportion of GPI cases to all cases of insanity was rising inexorably - if not dramatically.[9] The average percentage of general paralytics to total admissions between 1878 and 1882 was recorded by the annual Commissioners' Reports as 8%; a figure which had risen to 8.9% by the period 1888 to 1892. 'What conclusion can be drawn', asked Stewart, the asylum superintendent who analysed these figures, '... [other] than this, that general paralysis is increasing at a rate which is out of proportion to that which applies to other forms of insanity?'[10] One alternative conclusion was that the disease was being diagnosed with greater certainty - and this argument had clearly been applied for the early years of the century. The same could not be said, Stewart insisted, for the latter period: diagnostic ability could not have changed, he argued, over as short a space as fifteen years - particularly as there had been little change in the senior staff of British asylums. Besides, the increase had not applied equally to all classes of patient, but most strikingly to private male patients.

Despite an encouraging dip between 1890 and 1900, annual asylum admission figures continued to rise disproportionately in the years leading up to the First World War.[11] A new source of data also became available. For much of the nineteenth century the Registrar General's annual mortality statistics had offered only the broad categories of 'Insanity' and 'Paralysis' - under either of which heading GPI might have been listed. During the 1880's GPI was specified for the first time as belonging to the category of 'Insanity', but not until 1901 were

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[9] See Skae, 1859-60, p 886; Stewart, 1896.
[10] Stewart, 1896, p 762.
[11] From just over 1200 in 1900 to a peak of just over 1700 in 1912.

figures for the disease listed separately.[12] The mortality figures were regarded by contemporaries with caution; but it was acknowledged that GPI was sufficiently well-defined to take them seriously, and there were no major changes in post-mortem classification of the disorder at the beginning of the century. Somewhat surprisingly they showed no overall rise between 1900 and 1914 - remaining at an average of 2,250 per annum; and in fact death rates within asylums dropped slightly over the period. Nevertheless the perception that GPI was widening its domain dominated medical thought - a perception perhaps less to do with statistics than with the now stubborn identification of GPI as a disease of urban degeneration. Stewart, for example, used his interpretation of the figures to demonstrate rapid moral and physical decline, and an increasing tendency to racial decay.[13] Correspondingly in 1901 he related the short-lived small fall in GPI admissions to 'a tendency towards greater power of resistance and increased vitality...'; a happy consequence of greater alcoholic and sexual continence and more healthy outlets for energy - in particular the advent of cycling in towns![14]

Images of civilisation, degeneration, and the broadening domain of syphilis - as highlighted by the advent of the Wassermann test - provided Edwardian psychiatrists with the most compelling rationale for the rise of GPI. During the 1910's, however, more sophisticated immunological explanations appeared which focused attention upon the natural history of the syphilis organism and its relationship with the host. Multifactorial causal explanations for GPI were used well after the link with syphilis had been established; and until the 1910's such explanations posited a balance between syphilis - the 'seed' - and a nervous

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[12] See 75th Registrar General's Annual Report. PRO, Kew: Pp 1914-16, viii, part 3, p lxi.
[13] Stewart, 1896, p 777.

system weakened by the stresses of civilisation - the 'soil'. Syphilis might indeed appear on both sides of the equation: by insidiously weakening the nervous constitution in those cursed with acquired or congenital disease, and by directly exciting GPI, it could act as both soil and seed. Such multifactorialism provided a satisfactory explanation as to why only 3 to 5% of syphilitics went on to develop neurosyphilis.

But as experimental laboratory work upon syphilis accumulated, such problems came to be framed in more strictly immunological terms. The 'neurotropic' or 'dualist' hypothesis removed emphasis from the nervous soil, and provided an explanation for the syphilis-neurosyphilis link purely upon the basis of the seed. The hypothesis suggested that the syphilis organism appeared in two forms, or 'strains', causing two distinct varieties of disease: a 'dermatotropic' form which caused an acute illness with prominent skin and visceral lesions; and a 'neurotropic' form which caused a more chronic illness, resistant to treatment, with milder systemic symptoms but an affinity for the nervous system - leading to GPI and tabes. Sporadic pieces of clinical evidence supporting dualism regularly appeared in the literature from the turn of the century - for example observations of clusters of familial and conjugal GPI, and the common observation that a number of civilised countries such as Persia and China were experiencing widespread syphilis, yet practically no reported cases of general paralysis. The identification of the spirochaete, and its observation in the brains of general paralytics, allowed testing of the hypothesis to move to an experimental level. The idea was championed in America by Noguchi - who claimed that there were visible morphological differences between spirochaetes - and on the Continent by

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[14] R S Stewart, 'Decrease of GPI in England and Wales', J Ment Sci, 1901, 47, pp 41 - 48; p 47.

Levaditi and Marie - who supported it with animal experiments. The question of dualism was debated vigorously in the British literature from the 1910's to the 1930's, but evidence was usually regarded as inconclusive and the hypothesis never received widespread support. Those who took the 'unicist' line had plenty of counter-evidence. They argued that the two forms were not as clinically distinct as the dualists claimed, that Europeans who moved to Persia did themselves develop GPI, that until the spirochaete could be cultured little could be proven of its nature, and that clinical evidence was anecdotal and not statistically significant. A milder form of systemic syphilis was, they agreed, peculiarly related to GPI, but this was because mild disease was under-recognised, poorly treated, and so more likely to lead to chronic manifestations - not because it was related to a different strain of syphilis.[15] Fournier's nineteenth-century aphorism had recognised this fact: 'The milder the syphilis, the greater the tendency to tabes and general paralysis'.[16]

Neurotropism represented an extremely nominalist, bacteriological explanation of the identity of syphilis and GPI - an approach that perhaps already appeared anachronistic to many doctors by the early twentieth century, who could not discount the importance of the 'soil'. Its luke-warm reception corroborates the suggestion that the advent of microbiological thinking did not necessarily narrow

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[15] For discussions in the British literature, see G M Robertson, 'The Morison Lectures, 1913: General Paralysis of the Insane', J Ment Sci, 1913, 59, pp 185 - 221; p 211 - 212; Anon, 'The Specificity of the Syphilitic Organism in General Paresis', Lancet, May 14, 1921, p 1033; F W Mott, 'Modern Methods of Diagnosis and Treatment of the Nervous System', Lancet, Jan 6, 1923, pp 1- 4; P M Bigland, G A Watson, A D Bigland, 'General Paralysis and Somatic Syphilis', Lancet, Sept 20, 1924, pp 588 - 593; and E L Hutton, 'Recent Advances in the Aetiology and Treatment of Neurosyphilis', J Ment Sci, 1941, 87, pp 1 - 49; esp pp 3 - 11.

[16] Reiterated by Morris in 1912: 'In short ... the milder the syphilis - even to the degree of its having escaped the notice of the patient so that he may be quite unaware that he had ever had the disease - the greater is the tendency to tabes and general paralysis ...' H Morris, 'Observations on Syphilis', Lancet, Aug 24, 1912, pp 500 - 504; on p 502.

the perception of non-bacteriological dimensions to infectious disease. This has been demonstrated most extensively for the cases of TB and typhoid fever - although with particular emphasis upon the implications for public health strategies rather than for the development of immunological ideas.[17] Many American and some British authors accepted the compromise of 'acquired neurotropism'; an hypothesis, championed by Sicard, which suggested that spirochaetes which entered the nervous system gradually adapted, developing attributes which characterised the neurotropic form of syphilis.[18] Nevertheless by the 1940's the neurotropic theory was usually mentioned only in passing in text-books, if at all, and it was accepted that there was only slender evidence for the concept.

Neurotropism was used during this period to explain the static relationship between syphilis and GPI (including its geographical distribution) rather than the change in incidence of GPI over time.[19] In 1912, however, Frederick Mott offered an immunological explanation of GPI's apparent rise which was based upon a dynamic view of the history of syphilis itself. Syphilis had long been viewed in a historical light, primarily because of its apparently dramatic entry into Western

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[17] See, for example, L Bryder, Below the Magic Mountain (Oxford: Clarendon Press, 1988); F B Smith, who discusses the range of post-Koch aetiological theories of TB in The Retreat of Tuberculosis: 1850 - 1950 (London, New York, Sydney: Croom Helm, 1988); J W Leavitt, who considers public health responses to the carrier state in '"Typhoid Mary" Strikes Back: Bacteriological Theory and Practice in Early Twentieth-Century Public Health', Isis, 1992, 83, pp 608 - 629; M Pelling, 'Contagion/Germ Theory/Specificity', in Bynum and Porter, 1993, Vol I, pp 309 - 334; esp p 329. Some of these accounts place more emphasis upon the role of reversion to group (pace individual) public health strategies than upon developments in immunology in modifiying the extreme of biological determinism suggested by germ theory; the reciprocal influence of strategy and immunological theory, however, has not been clarified. Surprisingly, the standard histories of immunology make little reference to ideas of host resistance and seed/soil relationships and their implications for aetiological ideas.

[18] Supported in Britain, for example, by J F Smyth, 'The Relation of the Spirochaete Pallida to the Pathological Changes of Dementia Paralytica', J Ment Sci, 1928, 74, pp 687 - 708.

society during the fifteenth century, and ensuing debates over whether or not it was a new disease imported from America.[20] When attention first turned from the origin of syphilis to the question of its gradual change in nature through history is unclear. Accounts of medical geography and the acclimatisation of populations to disease in a static, spatial sense were common in early antiquity; but interest in diseases as historical entities subject to change, and in population acclimatisation in a dynamic, temporal sense did not become evident until the late eighteenth century.[21] During the nineteenth century syphilis was a common focus of this growing interest in the histories of diseases and the populations they attacked. Hirsch's huge late nineteenth-century survey noted the predictable reduction in virulence of syphilis in the years after it was introduced into a new population. His explanation was based solely upon human and environmental factors: poor public hygiene and uncurbed prostitution meant that the virus became attenuated with constant transmission.[22] But prior to this, by the 1870's, evolutionary theory was being applied to microbiological theory to provide new explanations of disease change over time, and of the inter-relationship between pathogenic

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[19] Although authors such as Hudson were developing hypotheses as early as the 1920's to suggest that related treponematoses had evolved from a single strain: see E H Hudson, Non-Venereal Syphilis: A Sociological and Medical Study of Bejel (Edinburgh: Livingstone, 1958).

[20] Quetel tells us that this debate was firmly in place by the beginning of the sixteenth century, and charts its progress until the early twentieth century when Sudhoff and Bloch took opposing views of the origin of syphilis: see Quetel, 1990, esp Ch 2.

[21] See, for example, Prichard who, in his Researches into the Physical History of Mankind (1836), wrote of acclimatisation of populations to disease through constitutional or acquired resistance; E Ackerknecht, History and Geography of the Most Important Diseases (New York, London: Hafner Publishing Co, 1965); D Brothwell, 'Disease, Micro-Evolution and Earlier Populations: An Important Bridge Between Medical History and Human Biology', in E Clarke (ed), Modern Methods in the History of Medicine (London: The Athlone Press, 1971), pp 112 - 134. J H Warner writes of theories of disease change in early nineteenth century Edinburgh, in which the nature of either the inflammatory fevers, or the human constitution, was claimed by some adherents of blood-letting to have altered over twenty years: see J Harley Warner, 'Therapeutic Explanations and the Edinburgh Bloodletting Controversy: Two Perspectives on the Medical Meaning of Science in the Mid-Nineteenth Century', Med Hist, 1980, 24, pp 241 - 258.

[22] A Hirsch, Handbook of Geographical and Historical Pathology, transl from German 2e by C Creighton (London: The New Sydenham Society, 1883), Vol II, p 59 et seq.

organisms and human populations. Scientists such as Lankester and Adami wrote of the evolution of biological variations in bacteria, and related these to the arrival of new diseases and their changing characteristics.[23] It was equally recognised that individuals with greater resisting powers might be naturally selected - so that diseases became less effective within populations as time passed. During the 1890's Archdall Reid offered a comprehensive account of the history of disease from the standpoint of Darwinian evolution - suggesting that the present evolution of man was chiefly shaped by the zymoses. Reid - in contrast to Adami - paid equal attention to 'soil' and 'seed' in his interpretation of acclimatisation: diseases stimulated the evolution of increasing powers of immunological resistance amongst populations; but microbes themselves evolved in response. The result was gradually decreasing virulence until equilibrium was attained between pathogen and host.[24] Such works generally made little mention of the later evolution of syphilis, usually concentrating upon its medieval appearance. By the early twentieth century, however, it was commonplace to speak of a change in syphilis from a florid infection to more insidious, latent forms - a fever diluted by time.[25]

It was this decline in virulence which Mott used in his own explanation for the rise of GPI.[26] The attenuation of syphilis had occurred, he argued, partly because natural selection had favoured those members of the population who were

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[23] See, for example, J G Adami, Medical Contributions to the Study of Evolution (London: Macmillan and Co, 1918), which contains essays from 1892 onwards.
[24] G Archdall Reid, The Present Evolution of Man (London: Chapman and Hall, 1896) and The Principles of Heredity (London: Chapman and Hall, 1905).
[25] See, for example, H Ellis, Studies in the Psychology of Sex . Vol 6: Sex in relation to society (Philadelphia: FA Davis Co, Publishers, 1920), p 320; H Rolleston, 'Changes in the Clinical Aspects of Disease', BMJ, Jan 15, 1927 (i), pp 87 - 91; p 88.

more immune to it; and partly because of prolonged mercury treatment. Mott was less impressed by the ensuing argument that a mild or latent syphilis was insufficiently treated and so more liable to lead to chronic manifestations such as GPI. Instead he suggested that in those individuals highly immune to syphilis, an excessive defensive reaction to the virus occurred which damaged the nerve cells. His theory was not exclusive: he also allowed a possible role for a neurotropic form of syphilis; and placed great emphasis upon degeneration of the neurones through the stressful effects of civilisation.[27] Primarily, however, his account explained the rise of chronic neurological forms in the context of the reduced virulence of syphilis - although it did not extend to the suggestion that GPI was an entirely new form of disease at the beginning of the nineteenth century. As well as drawing strongly upon modern immunological interpretations of disease, it also highlighted the dangers of half-hearted treatment of primary syphilis and lack of vigilance - at a time when the Wassermann test was revealing hidden syphilis everywhere. This naturalistic and historical approach to the rise of GPI was rather lost in later interpretations which placed more exclusive emphasis upon the deleterious effects of medical treatment. A variation of the theory, for example, suggested that the treatment of syphilis sterilised the systemic part of the patient, repressing the immunological reaction, and allowing the disease to run rampant in the nervous system.[28] This was the main explanation offered by the Royal

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[26] F W Mott, 'The Relation of Syphilis to Public Health, including Congenital Syphilis', Lancet, June 15, 1912, pp 1611-14. This is the first discussion of such a theory that I can find in the literature - although it is not clear whether it was original to Mott.

[27] See Chapter 3 above.

[28] See, for example, J E R McDonagh, 'Venereal Diseases as we see them today', The Practitioner, 1913, 91, pp 807 - 828; p 817.

Commission report of 1916 for the rise in neurosyphilis, despite better treatment for primary syphilis.[29]

GPI after the First World War

During the War years, figures for asylum admissions were not complete enough to study details of the incidence of GPI. Nevertheless, by 1920 it was clear that a dramatic drop had occurred in both the number of GPI-diagnosed admissions to asylums, and the reported annual deaths. Both trends continued steadily during the following years: from an average of 2250 between 1900 and 1914, the death rate had fallen to about 1750 by 1920 and to about 1150 by 1930; similarly, from a peak of 1700 in 1912, annual asylum admissions had fallen to 1300 by 1920 and to 1240 by 1930. Asylum death rates and statistics for life insurance companies corroborated this evidence.[30]

Treatment of neurosyphilis by malaria therapy had, of course, arrived too late to take credit for the decline. Instead, the majority of doctors explained the dramatic fall in terms of the success of diagnosis, prevention, and treatment of primary syphilis. On the one hand, it was argued, better education of the public was leading to prevention of the contraction of syphilis in the first place. On the other hand better education of doctors, more accurate methods of diagnosis, and more prompt and effective treatment was curing early cases of syphilis and preventing them from leading to neurosyphilis. Some suggested that intensive treatment of syphilis during World War I might have had the major effect; and a

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[29] Report of the Royal Commission, 1916.

[30] For reports of the fall see, for example, R N Ironside, 'On the Treatment of General Paralysis by Malaria Inoculation', Brit J Ven Dis, 1925, 1, 1, pp 58 - 63; T D Power, 'The Aetiology of GPI', J

number of contemporary and later accounts placed the decline firmly in the context of the Government's organised campaign against venereal disease - which hadn't, of course, begun in earnest until the early 1920's.[31] This assertion, as Mott pointed out in 1923, overlooked the long time-lag between syphilis infection and asylum admission or death due to neurosyphilis. When asked his opinion by the Board of Control in 1923, he noted that any decline in deaths must result from reductions in 'contracting' GPI about thirteen years previously - allowing for ten years before admission to an asylum and three years in the asylum before death.[32] The decrease therefore reflected reductions in syphilis infection from around 1907 due, he suggested, to greater detection by the Wassermann test (especially of those cases previously thought to be 'soft sore'), wider knowledge about venereal disease, and more effective early treatment. This latter point was open to question, since arsenicals had not been introduced until the early 1910's. Nevertheless GPI would, Mott predicted, continue to steadily diminish with greater knowledge, prevention, and early treatment of syphilis. His explanation of the fall in GPI became the official line of the Board of Control, and of most contemporary and later doctors.[33] In contrast with theories of the rise of GPI

Ment Sci, 1930, 76, pp 524-36; D Prentice, 'Syphilis in Mental Hospital Practice' and Discussion of the Royal Medico-Psychological Association, May 21, 1937, J Ment Sci, 1937, 83, pp 472-77; p 475.

[31] See for example D White, 'Thoughts on the Prevalence of Syphilis', BJVDs, 1925, 1,1, pp 136 - 145; R N Ironside, 'The Prophylaxis of Neurosyphilis', Brit J Ven Dis, 1927, 3, 4, pp 273-89; Rees, 1957. Later historians sometimes pushed the success story even further forward: see for example Kiple, 1993, p 1028: 'As a result of the introduction of antibiotic therapy, tertiary syphilis has almost disappeared.'

[32] Bond, letter to Mott, 3 March 1923. PRO, Kew: MH 53/ 539. The fact that the death rate started to decline 3 to 5 years after the decline in rate of admissions to asylums supported Mott's estimate.

[33] See for example E J Fitzgerald, 'Syphilis and "The Mental Treatment Act"', Brit J Ven Dis, 1934, 10, pp 117 - 137; pp 117 and 119; G M Robertson, 'The Prevention of Insanity', J Ment Sci, 1926, p 454; L G Brock, letter to W Jameson (Ministry of Health), 7 Dec 1942. PRO, Kew: MH 51 539: 'There is no reasonable doubt that the drop in GPI cases is due to better early treatment of syphilis.'

before the War, it concentrated exclusively upon the 'seed' of infection rather than the nervous or immunological 'soil'; and was a clear success story for modern measures against syphilis. Rather ironically, Mott's pre-war suggestion that ineffective treatment of syphilis had contributed to a rise in GPI (by reducing virulence) was now reversed: newly effective treatment was subsequently bringing about the decline of the tertiary disease.

Some doctors, however, could not accept that there had been significant advances in the management of syphilis as early as Mott claimed. Colonel Harrison, for example, questioned whether treatment during the first decade of the twentieth century had been any better than twenty or thirty years before; although he could offer no alternative explanation for the fall.[34] It is interesting that the 'soil' - although it was still invoked well into the 1930's to explain the relationship between syphilis and neurosyphilis - was only rarely used to explain the fall in GPI. The arguments of degeneration were not, it seems, readily inverted to argue for 'regeneration'; although a few did suggest the alternative that the nervous system was becoming more resistant, or that the immune system was becoming more effective. The psychiatrist Power, for example, was a traditionalist who called in 1930 for renewed attention to the soil - in the face of a 'Pasteurian tendency to concentrate upon the causative organism'.[35] Whilst acknowledging that the introduction of serological tests, laboratories and arsenical treatment might have had some effect on the decline in GPI, he placed more emphasis upon a putative decline in alcoholism (which he regarded as a direct

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[34] Quoted in G Robertson, letter to the Board of Control, 20 Feb 1923. PRO, Kew: MH 53, 539; also L W Harrison, 'Treatment of Syphilis: with special relation to its later manifestations', Lancet, Jan 6, 1923, pp 4 - 8; on p 5: 'I am not able to account for the great decrease in deaths from GPI

precursor of the disease), and better living conditions which had improved host resistance to infection.

There were doubts, too, about the figures themselves. After 1920 an increasing - though unquantifiable - number of patients were treated voluntarily in non-asylum sites. As late as the 1930's Nicol and Hutton believed that this figure might be substantial enough to skew the figures and falsely suggest that GPI was falling. Malaria therapy, they suggested, might be contributing to a falsely encouraging picture, since by lengthening patients' lives it was allowing them to die of causes other than GPI.[36] There were occasional suggestions, too, that changing criteria for defining GPI might have given the false impression of a drop in numbers. One such reference was based upon the apparent drop in alcoholism in recent years. GPI, it was argued, had been commonly mistaken for alcoholic insanity in the past; the real fall in alcoholism had therefore led to a fall in GPI as fewer 'false' cases were diagnosed.[37] The Wassermann test - and its possible role in changing the definition of GPI - entered into these debates less frequently than we might expect. If it were argued that the test had provided tighter, more accurate diagnoses of the disease, then it could be suggested that the extent of the fall had been exaggerated (since former levels were actually lower than claimed). On the other hand if the test had broadened the diagnosis - as a number of doctors claimed from the late 1920's - then the extent of the drop, it could be argued, might have been under-estimated. Such arguments did occasionally appear in the

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from 1919 to 1921'. Harrison also pointed out that the decline of GPI in women had occurred over a much more protracted period than that of men.

[35] Power, 1930, p 524.

[36] See W D Nicol, 'The Relation of Syphilis to Mental Disorder and the Treatment of GPI by Malaria', Brit J Ven Dis, 1933, 9, pp 219-29 ; Nicol and Hutton, J Ment Sci, 1935, p 814; Idem, 'Neurosyphilis: Its Treatment and Prophylaxis', Brit J Ven Dis, 1937, 13, pp 141 - 172; p 172.

[37] See 'General Paralysis: A Discussion ...', J Ment Sci, 1928, p 10.

literature, but did not form part of a major debate.[38] For most contemporary doctors medical advances provided an attractive rationale for the fall in GPI - just as degeneration theory had provided an attractive rationale for its previous rise.

Hare's Hypothesis

As the incidence of GPI fell steadily over the middle decades of the twentieth century, its interest as a current psychiatric problem correspondingly declined. In 1959, however, it re-emerged as a disease which still had intellectual and rhetorical uses for the psychiatry profession. In this year Edward Hare, consultant psychiatrist at the Bethlem Royal and Maudsley Hospital, published a detailed historical epidemiology of the disease which attributed its rise and fall to the natural processes of infection spread.[39] He suggested that GPI was caused by a neurotropic strain of the syphilis organism which had arisen by mutation in France at the beginning of the nineteenth century, spread throughout Europe and America, and finally died out when it had run its course. His account thus both revived the hypothesis of neurotropism, and applied it for the first time to a dynamic, rather than a static, account of the relationship between syphilis and GPI.

Hare's account was a painstaking piece of historical work; but it also corroborated his two most important professional concerns: the biological basis of psychiatric diseases, and their mutability over time. Hare was not, of course, unique in claiming that diseases evolved in response to the changing relationships between causative organism and host resistance. As I have noted, concepts of

[38] Power, 1930, for example, used this argument. Brander, of course, argued in 1928 that the diagnosis had been falsely broadened by the Wassermann test, but did not directly relate his argument to the changing epidemiology of GPI: see 'General Paralysis', 1928, pp 1 - 5.

change in disease were discussed throughout the nineteenth century and early twentieth centuries; and by the 1920's historical epidemiology, led by doctors such as Topley, Greenwood, and Ronald Ross was already an expanding field. Hare was, however, unique in applying this particular form of biological theory to psychiatric diseases; and in using his historical claims to support quite specific biological causal hypotheses.[40] Although he considered that perceived changes in mental illness might be due to non-biological - for example epistemological - factors, he was primarily committed to finding naturalistic explanations: 'The mutability of diseases', he wrote in 1981, 'has not attracted much attention from clinicians or historians; and after more than a hundred years, Darwin's ideas have still not made any great impact on medical thinking ... Diseases, like species, represent the balance of a process by which living organisms struggle to adjust to a continually changing environment ... And perhaps psychiatric diseases change more quickly than others because their expression is largely psychological and follows changing fashions in the mode of expressing mental distress.'[41] Such a rigorously biological stance might be expected from a psychiatrist at any period - but particularly one, trained in the Maudsley tradition, who began his work during the 1940's. Hare's use of history to support his stance, however, always demanded attention since he was unusual in eschewing the triumphalist approach to medical history, and in paying often exhaustive attention to detailed historical evidence.

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[39] For an account of Hare's career, see Obituary: 'Dr Edward Hare', Independent, 23 Dec, 1996, p 14.

[40] Earlier writers such as Hirsch and Creighton made little attempt to analyse the historical patterns of psychiatric disease, since they regarded the problems of definition and classification as too great.

[41] E Hare, 'The Two Manias: A Study of the Evolution of the Modern Concept of Mania', Brit J Psychiatry, 1981, 138, pp 89 - 99; on pp 94-5. Hare did not use the term 'disease evolution' in a

Hare's later work demonstrated his naturalistic approach particularly well. In his 1981 paper 'The Two Manias', for example, he noted that the non-dementing class of psychiatric disorders (now commonly recognised) was not delineated by German psychiatrists until the 1880's.[42] The traditional explanation for this lay in the main protagonist Kahlbaum's long-term work at one institution, which allowed him to classify symptoms on the basis of chronic outcome. Hare's explanation was that the severity and mortality of mental diseases in general had diminished over the past hundred years, so allowing less severe forms to manifest. This he attributed to McKeown's thesis that the health of populations had improved in general, mainly due to better nutrition and standards of hygiene: 'The effect of this was first apparent in the group of affective disorders, so allowing their identification late in the century as a distinct, non-dementing group. And we may then suppose that the same cause has continued to act, and that its effect has more recently become apparent in Kraepelin's dementia praecox group; and this would explain why we no longer see the 'profound dementia' which was still the characteristic end-state of the disorder in his day.'[43]

His next paper turned to the question of whether insanity rose in incidence during the nineteenth century; but his attention now turned to the role of cause rather than host resistance in disease change - 'seed' rather than 'soil'. Hare concluded that insanity did increase in incidence, and that much of this rise might be attributed to a slow infectious epidemic of schizophrenia which began around

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Darwinian sense as did, for example, Archdall Reid at the turn of the century, but simply as a reference to disease change with changing environmental conditions.

[42] Hare, 'The Two Manias ...', 1981.

[43] Hare, 1981, p 97.

1800 - a theme which he expanded in a paper published in 1988.[44] Again he backed up his argument with a wealth of historical detail, offering three main supporting observations: that there was no clear description of the disease before 1800; that insanity rose markedly thereafter; and that schizophrenia had persisted despite the low fertility of patients. Hare's papers demonstrated clearly the difficulties of weighing naturalistic against sociological or epistemological explanations of medical history, on the basis of often flimsy evidence. All too often - and Hare corroborates this - one is left with the conviction that only the historian's agenda (and in the majority of cases this is predominantly sociological) can finally decide what weight should be given to each: the debate between Hare and Scull concerning the apparent rise of nineteenth-century insanity demonstrates this difficulty.[45] Hare, however, at least gave serious consideration to non-biological explanations in most of his papers. It is true that, in his work on mania, he made little analysis of alternative interpretations: for example, that the intellectual and practical peculiarities of Kahlbaum's school allowed the new classification of mania to be elaborated. In later papers, however, he offered far more detailed assessments of the case for 'nosocomialists' (who claimed an apparent rise in insanity due to lower death rates in asylums, more complete registration, and more accurate detection) and addressed the problems of diagnostic classification in history. Whilst he continually stressed that, in the absence of hard data, he could offer only speculations, he succeeded in taking seriously the available evidence and in presenting a convincing case for his

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[44] Hare, 1983; 'Schizophrenia as a Recent Disease', Brit J Psychiatry, 1988, 153, pp 521-31. Hare referred here to the recency hypothesis of schizophrenia expounded by Torrey: E F Torrey, Schizophrenia and Civilization (London: Aronson, 1980).

historical arguments. His use of historical interpretation to support his commitment to a particular aetiological stance, however, remained obvious and interesting. Historical evidence was, to him, as valid as his more conventional scientific studies in support of the viral aetiology of schizophrenia.[46] He was quite frank in stating his hope of refuting the genetic basis of schizophrenia in the hope of 'finding [a hypothesis] less pessimistic than Bleuler's' which 'holds out no prospect of our ever being able to prevent schizophrenia by the elimination of some specific environmental cause.'[47] His alternative viral hypothesis, as well as bringing hope, also 'brings schizophrenia properly into the realm of medicine ...'[48] That the current consensus now regards evidence for a significant viral aetiology in schizophrenia as 'slight' is less relevant to this discussion than Hare's use of history.[49] Whilst all of his papers are appealing and quite possible solutions to much-debated historical problems, their most compelling feature is not the evidence presented but the ideological stance which underpins them: the conviction that predictable biological factors underlie mental diseases, which are therefore amenable to retrospective and objective analysis.

Hare's presentation of the epidemiological history of GPI was his earliest piece of historical work. His evidence for the theory of spread of a neurotropic virus was fourfold: the strong evidence that syphilis itself had changed in nature through history; the apparently new outbreak in Paris at the beginning of the nineteenth century; the pattern of prevalence and sex ratio in different countries;

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[45] Hare stressed an actual rise in the incidence of insanity; Scull stressed a number of social responses to insanity which led to an apparent rise in its incidence: Hare, 1983; Scull, 1989, Ch 9.
[46] See, for example, E H Hare, 'Season of birth in schizophrenia and neurosis', Am J Psychiatry, 1975, 132, pp 1168-71.
[47] Hare, 1983, p 450.
[48] Ibid, p 450.

the apparent change in clinical forms over 150 years; and the recent decline in prevalence. From the point of view of documentary detail his account was entirely convincing. He presented a strong case for GPI being truly a new disease at the beginning of the nineteenth century (pace the majority of his predecessors): arguing that there were no convincing descriptions before 1800; and, incidentally, showing a touching faith in the ability of eighteenth-century alienists not to overlook the striking symptoms of GPI.[50]

His analysis of the subsequent spread of GPI to countries outside France - appearing first in men and then in an increasing proportion of women - again is difficult to refute. To challenge the idea that the apparent rise had an actual basis, we would have to suggest that European doctors saw more GPI because of subjective factors: either because, as reports of the disease arrived from France and insane patients accumulated in asylums, they increasingly recognised it; or - to take a more audacious line - because they had an incentive to do so, since GPI demonstrated the professionally useful organic interpretation of mental disease. The strongest refutation of these suggestions comes from a close examination of asylum records. Descriptions of GPI from 1840 to the early 1900's stand out because of the rapid physical decline and death occurring in such patients. With the proviso that archival research tells us only a limited amount about the motives of diagnosing doctors, there is no evidence that patients with broader symptom patterns were drawn into this quite striking clinical pattern. Families, of course, might have been increasingly likely to send their general paralytic relatives to

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[49] See, for example, M Gelder, D Gath, R Mayou, Oxford Textbook of Psychiatry, 2e, (Oxford: OUP, 1989), p 300.

[50] Jacobowsky refuted the recency hypothesis by suggesting that GPI did exist before 1800, but that fevers such as smallpox carried off patients before it had a chance to manifest: an hypothesis

asylums during the nineteenth century - in line with arguments for insanity as a whole. But although this factor possibly played some part in the rise, the difficult nature and pitiful physical state of GPI patients would surely have rendered them much less likely to be cared for in the community in the first place; and Hare's argument that this 'soaking-up' effect would have anyway run its course quite rapidly after the introduction of asylums is valid.[51]

It is certainly true that doctors in most of Europe and America perceived a gradual decline in the classical grandiose form of insanity and a rise in the depressive or demented form in general paralytics over the course of a hundred years. In Britain this observation appears to have been less dramatic than in France or Germany, but was nonetheless well documented.[52] A change to milder, more varied and less stereotypical forms of the disease (paralleling the gradual avirulence of syphilis itself) was particularly apparent in the twentieth century, and appears quite obvious, for example, when examining asylum case-notes.[53] A number of sociological alternatives to Hare's naturalistic explanation of this phenomenon might be suggested. The change might, for example, be simply one of changed perception. Bayle's early delineation of the disease was clearly influential within and outside France, and his emphasis upon 'monomanie de grandeur' might have led alienists to stress this aspect of the newly-recognised

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which is extremely difficult to substantiate: B Jacobowsky, 'General Paresis and Civilisation' Acta Psychiatra Scandinavia, 1965, 41, pp 267-73.

[51] Hare, 1983.

[52] Conolly, for example, claimed in 1849 that delusions of grandeur were characteristic of the disease; by 1875, Skae and Clouston claimed that they were becoming less common. A number of twentieth century commentators noted that there had been a decline in grandiosity and an increase in demented forms over the past fifty years; see, for example, 'General Paralysis', 1928, p 76; Power, 1930.

[53] As, for example, in the case-notes of Horton asylum 1919 - 1950. Recent medical accounts of GPI corroborate this; for example J Towpic and E Nowakowska, 'Changing Patterns of Late Syphilis', Brit J Ven Dis, 1970, 46, pp 132-4, who explain such changes since the 1940's by the

disease during the early part of the nineteenth century. As GPI was diagnosed in greater numbers and with greater ease, a relaxation of diagnostic criteria might have taken place, so that cases with dementia were admitted more frequently, leading to the belief that this form was rising in incidence.[54] This point can again only be answered by the consistency of the 'core' description of GPI in clinical records - which emphasised characteristic physical symptoms, and did not specify the kind of insanity that accompanied the disorder.[55] And although the twentieth-century use of the Wassermann test might arguably have broadened the diagnosis to include less striking or atypical mental symptoms - a point that I would not over-stress - the long change over the course of the nineteenth century remains unexplained by it.

Similar problems apply to the question of the changing sex ratio. Hare noted a constant pattern of change in the European countries that he studied, with the male: female ratio beginning high and gradually reducing to a value of 4 to 2:1; a pattern which he attributed to the sexual behaviour of populations spreading the new infection. Britain showed this pattern less convincingly, and there were constant disagreements as to whether or not the ratio was falling; although most felt that women were increasingly succumbing to the disease.[56] The commonest contemporary explanation for a rise in women was that they were becoming

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widespread use of antibiotics leading to attenuated forms of the disease. Obviously, the explanation needs to go much further back.

[54] A Horton psychiatrist noted in 1905 the general opinion that the classical type of GPI was seen less than formerly; and pointed out that this might be due to a change in diagnostic criteria: H Baird, 'Statistical Observations on General Paresis', J Ment Sci, 1905, 51, pp 581-5.

[55] Nevertheless I have also discussed in Chapter 2 how ideas of accompanying insanity frequently reflected ideas about the aetiology of GPI, as well as conceptions of the pre-morbid personality of the sufferer; a further reminder that distinguishing 'chicken' from 'egg' in theories of disease is far from easy.

[56] See, for example, D White, 'Thoughts on the Prevalence of Syphilis', Brit J Ven Dis, 1925, 1, 2, pp 136-45.

increasingly prey to the causes of GPI - whether the stresses of civilisation during the nineteenth century or syphilis during the twentieth century. Once again, a perceptual change could conceivably be offered as an alternative to Hare's model. As the dementia form of GPI was more readily recognised, women - who were held to have this form more frequently in keeping with their pre-morbid character - might in turn have been more readily diagnosed.[57] Hare refuted the latter argument by pointing out that the Wassermann test did not reveal a sudden change in the number of women affected - an argument which holds even if the objectivity of the test (which Hare takes for granted) is not assumed.

Alternatives to Hare

Hare provided a satisfying and coherent biological account of the rise and fall of GPI. A number of alternative explanations, however, deserve consideration. Malaria therapy clearly cannot be used to explain the initial fall of GPI, which started five years or so before the new treatment was in common use; although curiously it was the inefficacy of malaria therapy, rather than the obvious time-lag, that Hare turned to when refuting the relevance of treatment advances to its decline. I have noted, however, that contemporaries sometimes suggested that malaria therapy might be exaggerating the fall in GPI deaths; since by lengthening the lives of patients in both asylums and the community, it allowed them to die of other causes.[58] I would agree with Hare that, even if two thirds of treated patients

[57] An anonymous commentator noted in 1907 that the proportion of female deaths from GPI had risen in Scotland in recent years; but, he continued, it was hard to tell how real this rise was, since the disease was 'now diagnosed with greater accuracy and frequency, especially in female patients ...': Anon, J Ment Sci 1907, 53, pp 151-5;on p 154. The latter point in the text could, of course, be argued the other way: as women developed the disease (in reality) more frequently, the demented form was increasingly recognised!

[58] Nicol and Hutton, 1937, p 172.

(roughly one third in the community and one third remaining in asylums) were recorded as dying from other causes, the reduction in deaths would still not be sufficient to account for the total reduction. Since this is assuming the extreme case, it is likely that malaria therapy - whilst causing a moderate accumulation of patients inside and outside asylums - exaggerated the extent of the fall in deaths to only a small degree.[59] Furthermore, the use of malaria therapy would not explain the continued fall in admissions to asylums - which paralleled that of the death rate. A further possible confounding factor - and one associated with malaria therapy - was the observation that an increasing number of patients were treated voluntarily in non-asylum sites after 1920.[60] However, although the number of patients treated outside asylums was notoriously difficult to estimate, it is again unlikely that it was large enough to produce more than a small exaggeration of the fall in admission figures. In addition, voluntary treatment appears not to have taken place in any large numbers until the beginning of the 1930's: ten years after the decline began.

The Registrar General's mortality figures for GPI, I have suggested, offer an adequately accurate guide to trends over time. Hardy makes the point that stigmatisation of syphilis itself probably led to a considerable degree of under-recording of mortality: in contrast, she adds, GPI was not closely linked to syphilis in the public eye until around the time of the First World War, and so was probably not under-recorded to the same degree.[61] This suggests the possibility that part of the fall in death rates recorded from 1920 onwards might be an

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[59] The Horton records suggest that the majority of patients treated with malaria were still recorded as dying from general paralysis.

[60] Nicol, as I have noted, believed that this figure might be substantial enough to challenge the impression that GPI was falling: Nicol and Hutton, 1935, p 814.

expression of under-recording following the link with syphilis. This is unlikely, however, to have been an important factor: general mortality rates, as I have noted, paralleled asylum figures which were presumably less confounded by fears of stigmatisation; also GPI was in fact linked with syphilis well before the 1920's, and was anyway a stigmatised disease before this link was made.

Hare made no mention of the most obvious alternative to his hypothesis: that the decline in GPI simply reflected a decline in the number of cases of primary syphilis developing into tertiary syphilis. Many contemporaries and later historians - as I have described - took this line; however, they placed it firmly within the context of medical advances. If we accept Mott's estimate of the average time elapsing between primary syphilis and the development of GPI, we would also have to accept his suggestion that medical improvements from around 1907 were sufficient to produce a significant drop in numbers. Yet this seems unlikely. Even granted that the Wassermann test did indeed detect more syphilis than ever before, it was not in common use until about 1910; and was not applied to significant numbers until after the First World War. By the 1920's, also, it had become clear that the seeds of neurosyphilis might be sown before the spinal Wassermann reaction became positive; and even Mott admitted that only a very small percentage of syphilitics had ever applied for treatment in the 'pre-Wassermann curable stage'.[62] Furthermore, the main problem in managing early syphilis remained - that of keeping track of patients and encouraging them to complete their course of treatment; and even after the clinics were introduced in 1920 (far too late to have affected the incidence of GPI), this problem appeared as

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[61] A Hardy, 'Death is the Cure of All Diseases: Using the General Registry Office Cause of Death Statistics for 1837 - 1920', Soc Hist Med, 1994, 7, pp 472-92; p 489.

insurmountable as ever. Treatment itself in the first decade of the twentieth century, as Harrison pointed out, was very little changed from that of thirty years previously; being based still upon mercury and potassium iodide. Salvarsan and neosalvarsan were in wide use only from about 1911 onwards; and even if we ignore the probability that they arrived a little too late to affect the incidence of GPI eight years later, their efficacy in preventing primary syphilis from leading to tertiary forms was seriously questioned at the time.

The changing incidence of syphilis itself has, of course, always been notoriously difficult to estimate; a point on which contemporary doctors and modern historians have concurred.[63] The Royal Commission on Venereal Diseases of 1916 admitted that any accurate estimate of the extent of the problem was impossible; in particular, the figures produced by the Registrar General were, they claimed, 'worthless' in this respect.[64] As a result, before the First World War there was a mixture of rather confused perceptions as to whether primary syphilis was on the rise or on the wane - perceptions that weren't necessarily drawn from the scanty evidence. The dubious mortality figures fell for all ages from 1900 onwards, and military statistics also seemed to suggest a decline: the incidence of syphilis, for example, in new recruits to the army and the navy was thought to be falling.[65] Some doctors claimed that they were seeing less of the disease; and a Lancet leader of 1913 noted that: 'It is usual to speak of syphilis as having very

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[62] Mott, letter to Bond, 3 March 1923. PRO, Kew: MH 51/ 539.
[63] See, for example, Anne Hardy's discussion of the inadequacy of statistical records for syphilis: 'The only certainty was the certainty of great understatement': Hardy, 1994, pp 489-90. The most obvious reasons were the social implications of the disease and its chronic, often latent, course.
[64] Royal Commission on Venereal Diseases, 1916, pp 21-3.
[65] From 1901 to 1910 recorded deaths at all ages up to 45 years fell; but deaths at ages above 45 years rose. According to Hardy, this was probably because doctors were increasingly able to recognise the tertiary manifestations, which they were now classifiying as syphilitic: Hardy, 1994.

much declined of late'.[66] Yet many interpreted the questionable evidence rather as an indication that syphilis was becoming more latent and insidious; and the Wassermann test, as I have discussed, was important in fuelling this perception. The dominant feeling was that syphilis was in fact rising, and posing more serious dangers than ever before. Morris, noting that the State's 'indifference' towards syphilis made statistical proof impossible, argued that it surely must be rising since pathetically half-hearted steps were being made to combat it.[67] Nonne made the same assumption of the German situation, arguing from recent changes in urban social conditions.[68] The Royal Commissioners' report concluded that, although military figures suggested that syphilis was falling, this was probably not true of the civilian population; and it tentatively concluded that 10% of the urban population had the disease. This confusion about the prevalence of syphilis before the War contrasted with the much firmer conviction of its decline from the 1920's onwards.[69]

Recent historical work suggests that military evidence could indeed be extrapolated to the general population, and that syphilis was probably declining well before the turn of the century. The idea that general improvements in nutrition might have had more effect upon disease attenuation than medical advances was well in place by the early 1950's.[70] McKeown's revisionist work from the mid-1970s denied far more explicitly that advances in medical science were responsible for the decline in mortality due to many major infectious diseases

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[66] Leader: 'Syphilis and the Responsibility of the State', Lancet, June 28, 1913, pp 1810-11.
[67] Morris, 1913. This lack of scientific accuracy was usually discussed in the context of the need for certification of syphilis.
[68] M Nonne, Syphilis und Nervensystem (Berlin: S Karger, 1902), p 543.
[69] See, for example, D Prentice, 'Syphilis in Mental Hospital Practice', J Ment Sci, 1937, 83, pp 472-7; A King '"These Dying Diseases": Venereology in Decline?', Lancet, 29 Mar, 1958, pp 651-7.

from the mid-nineteenth century.[71] In the ensuing debate some historians agreed with him that improvements in nutritional status could take most of the credit, whilst some restored prominence to other factors such as local public health measures.[72] Serious recent consideration of biological rather than social reasons for this decline has been left mainly to doctors (such as Hare) and epidemiologists interested in models of spread of infectious diseases.[73] Roy Anderson, for example, has used mathematical models of the transmission of infection to interpret epidemiological trends. He allows the importance of better nutrition and hygiene (and, in small part, medical advances) in explaining the fall in mortality over past centuries; but places more emphasis upon changes in genetic structure which have led to concomitant changes in parasite infectivity and host resistance. He notes - as did commentators eighty or more years before him - that parasites and host resistance evolve so that infections become avirulent over time.[74]

Debates such as these have rarely considered the historical pattern of syphilis: presumably partly because of the difficulty in evidence that I have mentioned; and partly because the venereal route of spread has made an analysis of epidemiological changes particularly complicated. A detailed paper by the venereologist J E Moore, however, appeared in 1951, which attempted to evaluate current public health measures against syphilis by placing them in the context of

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[70] See, for example, R Dubos, Louis Pasteur, Free Lance of Science (London: Gollancz, 1951).
[71] T McKeown, The Modern Rise of Population (London: Edward Arnold, 1976).
[72] S Szreter, 'The Importance of Social Intervention in Britain's Mortality Decline c 1850 - 1914: A Re-Interpretation of the Role of Public Health', Soc Hist Med, 1988, 1, pp 1 - 38; A Hardy, 1993. For overviews of these arguments, see R M Smith, 'Demography and Medicine' in Bynum and Porter, 1993, Vol II, pp 1663- 92; S J Kunitz, 'Medicine, Mortality, and Morbidity', in Ibid, pp 1693 - 1711; and K F Kiple (ed), The Cambridge World History of Human Disease (Cambridge: Cambridge University Press, 1993), esp pp 287-93.
[73] McKeown himself allowed little room for a natural decline in the virulence of infectious diseases.

statistics for the previous hundred years.[75] Moore concluded that syphilis had indeed declined throughout most of Western Europe and America from 1865 onwards, punctuated by transitory increases associated with war, political unrest, and population movements. He attributed the decline to a spontaneous change in host-parasite relationships; a significant part of which he linked to a gradual improvement in socio-economic conditions during the period and a gradual increase in population stability.[76] The decline, he concluded, continued after 1910 under the same influences, but was possibly accelerated by modern public health measures.[77] Since this paper a number of accounts have agreed that the incidence of syphilis fell more or less continuously in the West from the 1860's - although it is not always clear upon what basis these assertions are made, nor the suggested explanation.[78] Hardy also suggests that, notwithstanding the difficulty of interpreting the statistical evidence, any apparent rise in syphilis mortality figures from the end of the nineteenth century was probably in reality due to an extension of medical knowledge.[79]

If we postulate that, regardless of medical measures, syphilis was already declining by the end of the nineteenth century, then the fall of GPI at the beginning of the twentieth century might be explained on the basis of this. In the absence of accurate figures for syphilis this can be no more than an assumption, and it leaves

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[74] See, for example, R M Anderson and R M May (eds), Population Biology of Infectious Diseases (New York: Springer Verlag, 1982); R M Anderson and J M Thresh (eds), The Epidemiology and Ecology of Infectious Disease Agents (London: The Royal Society, 1988).

[75] J E Moore, 'An Evaluation of Public-Health Measures for the Control of Syphilis: An Epidemiological Study', Lancet, Mar 31, 1951, pp 699 - 711.

[76] Moore denied the impact of medical advances on this decline twenty years before McKeown did; in contrast to McKeown, however, he also allowed the importance of natural changes in virulence.

[77] For the period until 1910, Moore extrapolated from military to civilian figures; after this period civilian figures were more accessible, although they were still not conclusive.

[78] See A King and C Nicol, Venereal Diseases (London: Cassell, 1964); G W Csonka and J K Oates, Sexually Transmitted Diseases (London: Balliere Tindall, 1973); J Arrizabalaga, 'Syphilis' in Kiple, 1993, pp 1025-33.

a number of problems. First, there was a long time lag between the gradual decline of syphilis from the 1860's, and the more dramatic decline of GPI from the 1920's. Second, such an explanation still poses the problem of interpreting the sudden appearance of GPI during the 1820's: leaving us either with the classic explanation that it had not been recognised before; or a genuinely new appearance due to the change in nature of syphilis from acute to more chronic forms. Just as tertiary forms of the disease did not appear to arise until several hundred years after the arrival of syphilis in the West, it is possible that the 'parasyphilitic' forms did not appear until another hundred or so years again; and the changing nature of GPI would be explained by a continued attenuation of the syphilitic organism. This is simply an extension of Mott's theory that attenuation of syphilis (whether due to natural or therapeutic reasons) was leading to a precipitous increase in the incidence of GPI. The apparently rising proportion of women affected could - as I have discussed - be in some part due to increased recognition of the disease in women. Beyond this, it is harder to explain without using Hare's model of a separate infection - a point that is one of the strongest parts of his argument.

The rise and fall of GPI has been explained in a variety of ways useful to contemporaries: by diagnostic progress; by degenerative theory; and by therapeutic advances. Interpreted in this light, even a hypothesis of natural syphilis decline outlined above might be seen to serve a modern view of disease which down-plays medical triumphalism. Hare's account lies at an interesting chronological boundary. On the one hand it can be regarded as a retrospective explanation of disease epidemiology which is open to critique. Viewed in this way Hare made a convincing case for the recency of GPI, and for its subsequent clinical pattern. In

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[79] Hardy, 1994.

particular his account neatly explained all of the data that he observed: the appearance of GPI; its spread and rise; its changing nature and sex ratio; and its subsequent decline. But evaluating his hypothesis is a contentious project for the historian - particularly in the face of scanty and dubious data for both syphilis and GPI. The syphilitic spirochaete still cannot be cultured, and it is unlikely that science will come to the aid of the historian in evaluating the probability of the neurotropic hypothesis. More usefully perhaps, Hare's work can be viewed historically - as a final, rather fitting, episode in the history of GPI. Here, perhaps for the last time, psychiatry used the disorder as a persuasive paradigm - a paradigm in which mental diseases could be viewed as rigorous biological entities, with the same transhistorical status as any disease of mainstream medicine.

CONCLUSION

GPI can perhaps best be understood as the sum of several histories; but although it is convenient to consider these separately, their interdependencies are obvious. Triumphalism, most modern historians would agree, no longer has a valid place as one of these histories, and it is an approach that I have claimed to avoid through the course of my thesis. Nevertheless triumphalism makes its presence felt constantly in the context of one of the most prominent of my accounts: the ways in which GPI was put to use by the doctors who confronted it. From the moment that its appearance was first greeted as a conquest of modern clinical methods to its appropriation, a century and a half later, as the model for a hypothesis of biological change, GPI constantly epitomised hopes for progress and status in psychiatry. Through the vehicles of organicism, 'natural' classification, and neurophysiology it promised greater scientific credibility for nineteenth-century alienists. Through the vehicles of laboratory medicine and heroic physical treatments, it promised greater diagnostic and therapeutic credibility for their twentieth-century counterparts.

The rhetorical expression of these hopes was always obvious, whether it arose from working psychiatrists or - as was increasingly the case during the twentieth century - from regulatory bodies such as the Board of Control. But the translation of these hopes into practice was often less easy to assess. The gap between the two frequently represented the sheer elusiveness of mental disease; and the continued optimism of practitioners in the face of this often seems astonishing. This was

particularly the case during the nineteenth century, when despite their best efforts alienists' scientific words usually spoke far louder than their actions. Meanwhile, one senses their frustration as they watched neurologists set to work on their own group of patients with more tangible and impressive results. The gap was also, however, a factor of practical organisation. During the nineteenth century this was most obvious in the comparison between the working conditions of neurologists and asylum doctors. During the twentieth century it hinged more clearly upon the problem of co-ordinating with other organisations: whether the centralised laboratory; specialist and general hospitals; or venereal disease clinics. Increasingly, too, patients themselves were the frustrating factor. With the rise of voluntarism and an increasing emphasis upon disease before insanity, the antagonism of patients to interventions such as lumbar puncture was more and more a force to be reckoned with. Nevertheless in the twentieth century it is harder to unravel the relationship between rhetoric and practice - a difficulty which perhaps suggests that the gap was closing. The Wassermann test may not have revolutionised the diagnosis of GPI, nor paved the way for similar laboratory applications; but it made a tangible impact both within and outside the asylum, and quite probably stimulated the development of asylum laboratories. Similarly malaria may not have been the therapeutic miracle which psychiatrists hoped for; but it was applied in a limited way to early, non-certifiable disease, and made a largely positive impression upon the public and participating patients.

A second history of GPI - and one that is more elusive - concerns its construction by doctors. I have suggested that, amidst changing expectations and interpretations, psychiatrists kept their sights upon a coherent disease entity

characterised by core features. This means, for example, that I have assumed it is meaningful both to follow GPI's history as though it were a discrete entity, and to regard epidemiological patterns as broadly commensurable between different periods. I have not, however, given great attention to the details of classifying GPI; and in particular I have not tried to decide retrospectively whether our modern conception of the disorder tallies with diagnoses made in the past. Instead I have paid more attention to the different meanings and different emphases which doctors attached to the core disease - interpretations which arose both from raw perceptions of GPI and from changing interests and ideologies.

The core features were to a certain degree selected soon after the appearance of GPI. Most important, British alienists claimed that insanity - whether overt or not - was absolutely integral to the physical syndrome. As an extension of this, both their conception of muscular weakness as cerebral 'paresis' and their long disinterest in spinal symptoms served the claim that mental and physical symptoms were unified in the brain. Here, then, two histories of GPI became one: the very construction of the disease served the professional aim of physicalism, and informed the relationship of alienists with general physicians, neurologists, and lawyers.

Accepting GPI as 'parasyphilis' during the late nineteenth century required, I have suggested, a major reconception in doctors' views of syphilis itself. There are a number of reasons why the reconception should have been possible at this particular time. Pathological work through the nineteenth century had uncovered more and more examples of remote tertiary manifestations of syphilis. This, together with the progressive entry of the disease into the public and social arena, was increasingly

highlighting its reputation as insidious and dissembling. The addition of the parasyphilitic diseases to such remote effects required, however, a turn away from pathological and clinical causal evidence to statistical evidence. Fournier, as spokesman for this revolution, made two persuasive statements. First, he presented statistical evidence as valid despite its contradiction with more traditional evidence; and late nineteenth-century doctors appear to have been ready to accept this principle. Second, he presented parasyphilis as part of a broad expansion of the domain of syphilis - notably through the congenital disease - which mirrored his own perception of the expansion of venereology. That British psychiatrists did not initially seize upon the link suggests that their caution in reconceptualising syphilis outweighed any uses that parasyphilis - as a thoroughly organic disorder - might have had for specialty claims. The conspicuous lack of specialty ambition amongst venereologists must also go some way to explain this.

As important as medical and specialist concerns in making the link, however, was the appropriation of degeneration theory towards the turn of the century. Ideas of GPI, mental dissolution, sexual excess, and syphilis converged easily within this framework, despite the common understanding that GPI rarely had a hereditary element. Until the first decade of the twentieth century, then, 'parasyphilis' did not represent the move to a specific theory of GPI. Rather, true to the stance of contemporary psychiatrists, it represented a multifactorial theory with strong degenerationist overtones - overtones which applied equally to Mott's stricter pathological conception of the disease.

The laboratory - represented by the Wassermann test - was clearly regarded as more authoritative evidence than statistics in forging the GPI-syphilis link, as demonstrated by the rapid change in terminology from 'parasyphilis' to the rigorously aetiological 'neurosyphilis'. Neurosyphilis embodied a more specific theory of the disease which coincided, in the years leading up to the First World War, with a loss of interest in degenerative explanations as a whole. Thus whilst multifactorialism would retain a place in the causal explanation of GPI, it would increasingly be expressed in the stricter terms of immunology. The reconception of GPI by laboratory medicine in aetiological terms is clear. What is less clear is how thoroughly this was worked out in the diagnostic conception of the disease. The test was clearly imported wholesale into the culture of GPI, and in particular changed its chronological dimensions by leading doctors to scrutinise very early disease in the pre-asylum stage - although not to the extent that rhetoric would imply. We can speculate that this had a profound effect upon how patients themselves experienced their illness. On the other hand, psychiatrists - like their general medical colleagues - retained a certain suspicion of the laboratory even whilst they used it rhetorically. Clinical acumen remained important to them, and it was rarely the case that they used the Wassermann test indiscriminately.

A further construction of GPI related to the perceived character of the patient and his insanity. I have argued that a dominant image of the general paralytic during the nineteenth century was one of vigorous, albeit uncontrolled, masculinity. This image bore a strong relationship to epidemiological observations - a relationship which was arguably reciprocal to some degree. It was also mirrored by the dominant

perception of the associated insanity: exuberant, grandiose delusions in which ideas of wealth and excess played a large part. As the century progressed, this image was opposed increasingly by that of the degenerate male - although the sympathetic reading, I suggest, remained strong. In parallel, doctors increasingly emphasised progressive dementia rather than florid delusions of grandeur; and the shift towards this more 'womanly' form of madness mirrored the perceived rise of the disease in intemperate, working class females. Just as the laboratory fostered scrutiny of the neurosyphilitic earlier and earlier in the course of his disease, so dementia suggested a more chronic course during its latter stages. With the advent of malaria therapy this development was complete: the patient, diagnosed and possibly treated before his insanity had become overt, might equally now spend many years of his life outside the asylum as a chronically demented invalid.

A third history of GPI - and one which is only obliquely told in this account - concerns the experience of the patient himself. My sources have been almost without exception produced by doctors; and they thus tell only partial truths about the disease. If we concede, however, that even accounts written by professionals might give some insight into the identity of their charges, it is possible to discern patients themselves moving ever more into the foreground. During the nineteenth century asylum notes recorded what were considered to be relevant aspects of patients' pasts. Delusions themselves - usually recorded in far more detail - often seemed, too, to reflect their lives and preoccupations. Not until the twentieth century, however, did patients start to visibly impinge upon their own disease: as, for example, when they refused lumbar punctures, or missed their appointments at the VD clinic. Malaria treatment drew

attention to patient autonomy more clearly than ever before. Certainly in pre-war Britain psychiatrists were largely free to apply their therapeutic experiment to patients who were often uninformed or uninformable. Patients, however, increasingly appeared to interact with their doctors, to express their dissatisfaction or misgivings, and to make limited decisions about their management.

Finally, it is the biological history of GPI - one which I have not entered into - that offers the starkest picture of the psychiatrist's continued frustration. Despite the intense attention given to the disorder by alienists; despite their varied responses, interpretations and exploitations, they were largely helpless as they watched the progressive accumulation of general paralytics in asylums. Then, as they began to apply to it the tools of laboratory science and dramatic heroic treatments, it seemed suddenly that they were pushing at an open door: GPI was now already a disease in decline. Medical measures against early syphilis were an attractive explanation for this. For many, however, the suggestion did not seem convincing; and besides, it was not a conquest that psychiatrists themselves could lay claim to. Although Hare's picture of the history of the disorder still served his profession's aims, ironically its message was disheartening: ever subject to the strenuous efforts and gestures of asylum doctors, GPI ran its inexorable course regardless.

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